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THE DISABLING DISEASES OF CHILDHOOD

THEIR CHARACTERISTICS AND MEDICAL CARE AS OBSERVED IN 500,000 CHILDREN IN EIGHTY-THREE CITIES CANVASSED IN THE NATIONAL HEALTH SURVEY OF 1935-1936

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The characteristic diseases of childhood are infectious. The supervision of water supplies, the sanitary disposal of sewage and the pasteurization of milk contribute to the control of certain of these diseases. By and large, however, the contact-borne infections present the major problem in the control of communicable disease today, and by establishing the diagnosis and reporting the case the physician takes the first step toward an effective program of prevention. Protection from certain communicable diseases may be secured by the creation of artificial immunity. Periodic medical supervision of well children is a valuable measure for the general promotion of child health. However essential, the development of these preventive health services must not be permitted to obscure the importance of medical care of the sick child as a means of protecting both the sick and the well.

Medical care of the sick child fills the important function of promoting recovery and reducing the incidence of sequelae which immediately or ultimately impair health. On the average, a high recovery rate is characteristic of the diseases of childhood, except in the period of infancy; but deaths of older children from causes to some degree preventable are by no means negligible. In the three year period 1933 through 1935, an average of 51 per cent of all deaths of children between 1 and 15 years of age were due to the infectious and parasitic diseases, pneumonia, and diarrhea and enteritis. An average of 23,000 deaths of children of these ages were caused yearly by diseases in the infectious and parasitic group, 10,746 by pneumonia, all forms, and 5,458 by diarrhea and enteritis. These deaths measure in part the result of lack of medical care and of delay in summoning medical aid beyond the point at which treatment is effective.

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The results of the National Health Survey present a general view of the medical and nursing care of disabling illness received in a twelve month period by over 500,000 children in eighty-three representative [1] urban communities in eighteen states. The survey employed the method of the house to house canvass, the information concerning the social and economic characteristics of the family and its records of illness and medical care in the survey year being obtained from a lay informant, usually the housewife. The canvass was made in large, medium-sized and small cities, and the results thus permit an examination of the effect of urbanization on the amount and nature of medical services for the disabling diseases of children. The records relating to annual family income and relief status in the survey year make possible the comparison of the medical care of children in different economic groups. The results of this survey of disabling illness in childhood form the subject of the present report. As an introduction to the consideration of the receipt of medical care, the characteristics and leading causes of disabling illness in childhood as observed in the survey will be discussed briefly.

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1. A list of the surveyed cities is given by Perrott, G. St. J.; Tibbitts, C., and Britten, R. H.: The National Health Survey: Scope and Method of the Nation-Wide Family Canvass of Sickness in Relation to Its Social and Economic Setting [Appendix B], Pub. Health Rep. **54**:1663, 1939.

A reproduction of the survey schedule was published in The New Health Survey, Government Services Dept., J. A. M. A. **105**:1127 (Oct. 5) 1935.

The sampling procedure consisted of a random selection of districts to be canvassed within each city, the districts used being those outlined for the enumeration of the population in the Federal Census of 1930. Districts containing approximately equivalent units of population were obtained by arbitrary division of the census enumeration districts having a population in excess of 1,000. The number of such districts to be surveyed was determined by the number of surveyed families required to give a sample adequately representing the given city and sufficient to produce an urban sample representative of all regions of the country and, within the limitations of the survey, balanced in respect to size of the cities included. A complete canvass was made of the districts selected in this manner. For a complete description of the sampling procedure, see the publication first mentioned in this footnote.

The distribution of the surveyed population by geographic area agrees closely with that of the total urban population as enumerated in the Federal Census of 1930. The distribution by population class of the city of residence is necessarily somewhat less representative, 74 per cent of the surveyed population being drawn from cities with population of 100,000 and over as compared with 52 per cent for the total urban population in 1930. For the cities of 25,000 to 100,000 the corresponding figures were as follows: health survey, 14 per cent; census of 1930, 19 per cent. For cities of less than 25,000 population the figures were: health survey, 12 per cent; census of 1930, 29 per cent.

CHARACTERISTICS OF DISABLING ILLNESS IN A TWELVE MONTH PERIOD

Frequency.—The results of the National Health Survey indicate that among white urban children the period included in the first ten years of life, exclusive of infancy, is characterized by a high frequency of disabling illness which is not again approximated until the period of old age. In this survey, a disabling illness was defined as a disease, injury or permanent gross impairment which had caused disability for at least seven consecutive days in a twelve month period.[2] A high frequency of illness among children under 10 years of age has been observed also in earlier surveys, in which a more inclusive definition of illness was adopted.[3]

The frequency rates of disabling illness according to age as observed in the survey are shown in the appendix to table 1; chart 1 shows graphically the age trend in the frequency rates. In childhood, exclusive of the ages under 1 year, the maximum incidence of disabling illness occurs in the age period 5 to 9 years and is followed in order by the frequency rates for the preschool period (1 to 4 years) and for late childhood (10 to 14 years). The frequency rates shown here describe the incidence of disabling illness in white urban children; characteristic differences have been observed in the rates for Negro children [4] and for children in rural areas.[5]

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2. Disability is used in the sense of inability to pursue the usual activities, i. e., play by the preschool child, school attendance by children of school age and gainful or other work by adults. Certain exceptions to the definition of disabling illness were made: Records of all confinements, hospital cases and deaths were taken without limitation as to the duration of disability. An additional exception was made in the enumeration of chronic diseases and permanent gross impairments, which were recorded without reference to the existence or duration of disability.

The diagnoses of illnesses having multiple causes were listed by the enumerator of the canvass in order of their importance as causes of incapacity. In the subsequent coding of these records, the primary, as distinguished from the contributory, cause of illness was taken as the condition which had caused the longest period of disability. An illness due to multiple causes was considered as a single *illness,* but data concerning the several causes were coded separately so that all *cases* of a given disease could be segregated, whether the condition was designated as the sole, the primary or a contributory cause of the illness.

3. (*a*) Sydenstricker, E.: The Age Curve of Illness; Hagerstown Morbidity Studies, Pub. Health Rep. **42**:1565, 1927. (*b*) Falk, I. S.; Klem, M. C., and Sinai, N.: The Incidence of Illness and the Receipt and Costs of Medical Care Among Representative Families, Publication 26, Committee on the Costs of Medical Care, Chicago, University of Chicago Press, 1933. (*c*) Collins, S. D.: A General View of the Causes of Illness and Death at Specific Ages, Pub. Health Rep. **50**:237, 1935.

4. Holland, D. F., and Perrott, G. St. J.: Health of the Negro, Quart. Bull. Milbank Mem. Fund **16**:5, 1938.

(Footnotes continued on next page)

The method of the present survey imposes certain limitations on the definition of the characteristics of disabling illness among infants under 1 year of age. The disabling illnesses recorded in the survey were those occurring in the twelve months prior to the date of the canvass. Infants under 1 year of age as of the survey date were the survivors of births occurring in the preceding twelve months and were therefore exposed to the risk of illness for periods varying from less than a month to twelve months. In this respect, the illness experience of infants under 1 year of age differs from that of members of the population at the ages of 1 year and over, who, with the exception of persons dying in the survey

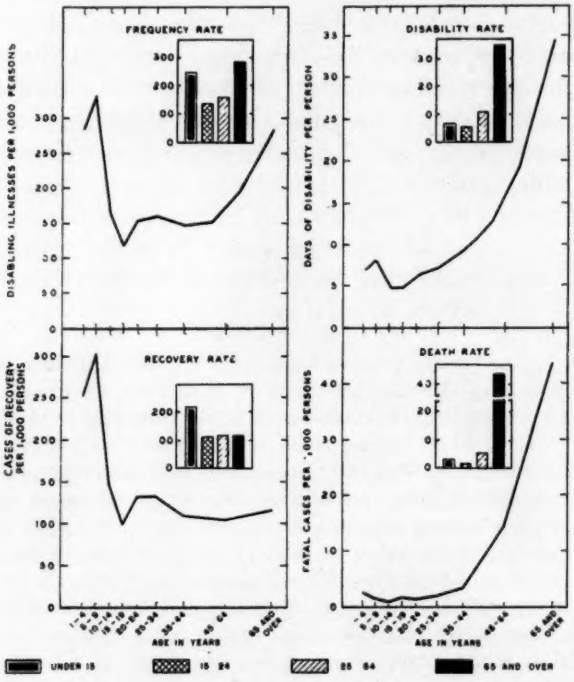

Chart 1.—Frequency, disability and recovery rates of illness disabling for seven consecutive days or longer, and the observed death rate based on all fatal cases in a twelve month period, by age, for 280,073 white persons in eight large cities canvassed in 1935-1936. The rates for infants under 1 year of age are not shown, since the period of exposure was less than twelve months.

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5. Preliminary study of disabling illness among rural children in three states canvassed in the National Health Survey. See also Godfrey, E. S., Jr.: The Age Distribution of Communicable Disease According to Size of Community, Am. J. Pub. Health **18**:616, 1928 and Fales, W. T.: The Age Distribution of Whooping Cough, Measles, Chicken-Pox, Scarlet Fever and Diphtheria in Various Areas in the United States, Am. J. Hyg. **8**:759, 1928.

year, were exposed to the risk of illness for twelve months. The conversion of the various measures of illness among infants under 1 year of age to a twelve month basis involves certain assumptions concerning the incidence rate of illness at specific months of age in the first year which cannot be verified on the basis of existing morbidity data. Future analysis of the results of the National Health Survey may contribute information on this point. The present report adopts the practice of expressing the *observed* rate of illness among infants under 1 year of age per thousand live births. Such a rate permits certain internal comparisons within the period of infancy, but its absolute magnitude in relation to the rates observed at subsequent ages is without significance.[6]

Severity.—The effect of the high frequency of illness on child health is ameliorated by the low mean duration and high recovery rate which characterize illness in childhood except during infancy. The severity rate, i. e., the mean duration of illness, was found to be lowest in childhood (appendix to table 1). As a result, the amount of disability accruing from the illnesses of childhood was disproportionately low with reference to the high frequency of illness at this period. This may be observed by a comparison of the frequency and disability rates plotted in chart 1. At specific ages of childhood, the relative variations in the disability and frequency rates were similar, the maximum point in both rates in the period of childhood occurring between the ages of 5 and 9 years. Children of these ages (including both the sick and the well) experienced eight days of disability per capita in the twelve month period from illness which involved a minimum of seven consecutive days of disability. The disability rate during these ages exceeded all subsequent rates up to the period 35 to 44 years.

The severity of illness may be evaluated also by a consideration of the probability of its termination in recovery or death. The recovery rate in childhood, exclusive of the first year, was found to be high compared with the rate in the adult period; and the death rate[7] is low

6. The only published data available on the incidence of illness among infants in a representative population are those obtained in the survey of the Committee on the Costs of Medical Care, reported by Falk, Klem and Sinai.[3b] These authors showed the incidence rate of illness, disabling and nondisabling, among (1) infants born during the survey year and (2) infants under 1 year of age at the beginning of the twelve month period of observation. The exposure of the second group comprises parts of the first and second years of life.

7. The death rates observed in the survey are lower than rates based on registered deaths; this deficiency results from the method employed (the house to house canvass), which does not obtain complete reporting of deaths. Previous surveys conducted by this method have shown a similar discrepancy. However, a correction for unreported deaths would not alter significantly the relative magnitude of the recovery and death rates.

compared with that of adults over 25 years of age. The nature of the age variation in these rates may be observed in the appendix to table 1 and in the two lower graphs of chart 1; the rates are based on the experience of surveyed persons in eight large cities. The appendix to table 1 includes also the frequency rates of incomplete cases, i. e., cases of persons sick on the day of the canvass who had been disabled for a minimum of seven days. It will be noted that illnesses which were not terminated at the date of the survey formed a lower proportion of all disabling illnesses in childhood than in adult life.

THE DISABLING DISEASES OF CHILDREN

Diseases Classified in Major Groups.—Eight in 10 disabling illnesses among children under 15 years of age observed in this survey were due to the acute communicable diseases or to the acute diseases of the respiratory tract, including influenza, tonsillitis, colds, pneumonia and bronchitis. Among these 8 cases, about 5 were cases of acute communicable diseases of childhood, and 3, of acute disease of the respiratory tract.[8] These ratios are based on the frequency rates of disabling illness classified by cause in major groups, shown in table 1.

The acute communicable diseases are uniquely characteristic of childhood. In this respect the incidence of acute diseases of the respiratory tract shows an important difference, reaching a maximum

8. For the purpose of a broad classification of the causes of disabling illness in childhood, four groups of diseases having certain common characteristics have been used. By excluding influenza, tuberculosis and specific infections of the intestinal tract from the specific infectious diseases, a new communicable group has been established which comprises mainly the common communicable diseases of childhood: measles, mumps, chickenpox, whooping cough, scarlet fever and diphtheria. Influenza has been combined with the diseases of the nose, throat and lungs (except tuberculosis of the respiratory tract) to form the respiratory group, which in childhood includes largely acute diseases: tonsillitis, colds, pneumonia and bronchitis, in addition to influenza. The specific infectious diseases of the intestinal tract have been combined with other diseases of the digestive system to form the digestive group. This classification comprises the following disorders: appendicitis, indigestion, biliousness, diarrhea and enteritis, ulcer of the stomach or duodenum and diseases of the gallbladder or liver. Finally, tuberculosis in all forms; nervous and mental disease or defect; cancer; rheumatism; diabetes; cerebral hemorrhage and paralysis due to other causes; diseases of the heart, arteriosclerosis and high blood pressure and other diseases of the circulatory system, exclusive of hemorrhoids and varicose veins; nephritis and other nonvenereal diseases of the genitourinary system, exclusive of circumcision and diseases of the female genital organs, have been combined under the group of major chronic diseases. By definition, certain chronic diseases of the respiratory and digestive systems are included, respectively, in the respiratory and the digestive groups; however, the incidence of these chronic diseases is relatively low in childhood, and among children under 15 years of age the respiratory and digestive groups as used here comprise chiefly acute diseases.

in childhood but maintaining a relatively high level in the adult period. The nature of the age variation in the frequency of disabling illness due to the major groups of diseases may be observed in chart 2.

TABLE 1.—*Frequency, Severity and Disability Rates of Disabling Illness Classified by Age and Sole or Primary Cause Among 2,152,740 White Persons in Eighty-Three Cities in a Twelve Month Period* *

Age Period, Yr.	All Causes	Selected Diseases									
		Communicable Diseases †	Diseases of Respiratory System ‡	Major Chronic Diseases and Orthopedic Impairments					Diseases of Digestive System ¶	All Other Diseases	Accidents
				Total	Tuberculosis, All Forms	Nervous and Mental Diseases §	Other Major Chronic Diseases ‖	Orthopedic Impairments			
	Frequency Rate (Disabling Illnesses per 1,000 Persons)										
All ages #.....	171.4	30.3	50.0	29.7	1.3	5.5	20.0	2.9	12.6	33.2	15.6
Under 15.....	224.6	105.4	74.2	9.1	0.5	2.6	4.8	1.2	7.5	17.5	10.9
15-24.....	128.8	12.4	37.1	11.9	1.5	3.9	5.0	1.5	13.7	40.6	13.1
25-64.....	149.6	5.0	42.2	33.5	1.7	6.8	22.1	2.9	13.8	38.0	17.2
65 and over.....	275.6	2.3	58.6	134.7	0.9	9.9	109.3	14.6	19.6	33.2	27.1
	Disability Rate (Days of Disability per Person)										
All ages #.....	9.83	0.70	1.15	4.69	0.32	1.03	2.45	0.89	0.74	1.79	0.75
Under 15.....	5.93	2.24	1.26	1.12	0.09	0.43	0.32	0.28	0.23	0.72	0.36
15-24.....	5.31	0.28	0.67	1.96	0.34	0.81	0.44	0.38	0.53	1.35	0.51
25-64.....	10.26	0.19	1.11	5.14	0.43	1.28	2.56	0.87	0.91	2.03	0.88
65 and over.....	35.44	0.15	2.54	23.53	0.20	1.99	16.26	5.08	1.96	5.28	1.97
	Severity Rate (Days of Disability per Disabling Illness)										
All ages #.....	57	23	23	158	243	190	123	306	59	54	48
Under 15.....	26	21	17	124	184	169	66	235	30	41	33
15-24.....	41	22	18	166	232	206	89	251	39	33	39
25-64.....	69	38	26	154	256	188	116	305	66	54	51
65 and over.....	129	65	43	175	221	203	149	347	100	160	73

* Illness disabling for seven consecutive days or longer in a twelve month period. All confinements, fatal illnesses and illnesses treated in a hospital are included without reference to the duration of disability. The 2,152,740 white persons are exclusive of persons in families for which income was reported as unknown.

† Including chiefly the communicable diseases of childhood: measles, mumps, chickenpox, whooping cough, scarlet fever and diphtheria.

‡ Including influenza, pneumonia, cold, bronchitis, tonsillitis, pleurisy, sinusitis, asthma, hay fever and other diseases of the respiratory system except tuberculosis of the respiratory tract.

§ Including mental defects.

‖ Including cancer; rheumatism; diabetes; cerebral hemorrhage and other forms of paralysis; diseases of the heart, arteriosclerosis and high blood pressure, and other diseases of the circulatory system exclusive of hemorrhoids and varicose veins; nephritis and other nonvenereal diseases of the genitourinary system exclusive of diseases of the female genital organs.

¶ Including appendicitis, indigestion, biliousness, diarrhea and enteritis, ulcer of the stomach or duodenum, diseases of the gallbladder or liver and other diseases of the digestive system.

Rates for all ages are based on the total cases and total population of known ages only.

The communicable diseases and diseases of the respiratory tract likewise accounted for over half of the disability of children under 15 years of age in the survey year, the communicable diseases disabling each child on the average about two days and the acute respiratory diseases about one day. The major chronic diseases (including orthopedic impairments) accounted for approximately one day of disability per

child; however, only 9 disabling illnesses due to these causes occurred in every 1,000 children, as compared with frequency rates of 105 per thousand for the communicable diseases and 74 per thousand for diseases of the respiratory system.

APPENDIX TO TABLE 1.—*Age Variation in Frequency Rates of Disabling Illness* in a Sample of 2,152,740 White Persons† in Eighty-Three Cities and in Frequency Rates of Illness* According to Termination, Severity Rate and Disability Rate Among 280,073 White Persons in Eight Cities‡ in a Twelve Month Period*

Age Period, Yr.	Sample of 83 Cities: Frequency Rate of Disabling Illness,* Total§	8 Cities: Frequency Rate of Disabling Illness* with Specified Termination				Disability Rate	Severity Rate
		Total§	Recovery	Death	Incomplete‖		
	Rate per 1,000 Persons (At Ages Under 1 Year, per 1,000 Live Births)					Days of Disability per Person	Days of Disability per Disabling Illness*
All ages¶	170	184	139	6.65	38.1	10.4	57
Under 15	225	249	220	3.16	25.6	6.4	26
Under 1#	120	134	74	30.37	29.8	3.6	27
1-4	251	284	253	2.87	27.6	6.9	24
5-9	305	336	304	1.34	30.5	8.2	24
10-14	153	169	148	0.92	19.4	4.9	29
15-19	107	117	98	1.48	17.0	4.7	40
20-24	148	154	132	1.45	20.6	6.4	42
25-34	151	161	133	1.73	26.0	7.3	46
35-44	136	147	109	3.40	34.6	9.3	63
45-64	155	170	104	10.45	55.3	15.2	89
65 and over	273	283	115	43.74	124.9	34.8	123

* Illness disabling for seven consecutive days or longer in a twelve month period. All confinements, fatal cases and hospital cases are included without reference to the duration of disability; sole or primary causes only are tabulated.

† Exclusive of persons of unknown age or unknown income.

‡ The eight cities include Atlanta; Cincinnati; Dallas, Texas; Fall River, Mass.; Newark, N. J.; Oakland, Calif.; St. Paul and Seattle.

§ Disabling illnesses (as defined in the first footnote) per thousand persons; this rate represents the sum of the rates for fatal and incomplete cases and for those in which recovery occurred.

‖ I. e., disabled on the date of the canvass after an illness causing incapacity for at least seven consecutive days.

¶ The rates for persons of all ages in eight cities include a small number of cases and persons of unknown age; the rate for persons of all ages in eighty-three cities is exclusive of cases and persons of unknown age.

In the period under 1 year, the frequency rates are computed per thousand live births and the disability rate per live birth.

Broadly considered, the groups of acute communicable diseases and diseases of the respiratory tract thus include the major diseases of childhood. The individual child suffering from heart disease, tuberculosis or crippling impairment presents medical and social problems of the first importance; on the average, however, these diseases account for relatively little disability in childhood.

The incidence of the communicable diseases was found to be notably higher among children in the small cities (population under 25,000)

than in the intermediate (population 25,000 to 100,000) and the large cities (population 100,000 and over), as is shown in table 2. The excess is not attributable to epidemics occurring in the small cities of a single region, since it is apparent in the rates of small cities in each geographic area. Furthermore, the excess in the small-city rates is marked in each income class. The frequency rates in table 3, classified by income, indicate that illness due to the communicable diseases was 82 per cent more frequent in children of relief families in the small

TABLE 2.—*Frequency Rate and Frequency Index of Disabling Illness* According to Sole or Primary Cause Among 518,767 White Children Under Fifteen Years of Age in Eighty-Three Cities Classified by Geographic Area and Size in a Twelve Month Period*

Area and Population Class of Surveyed City	All Causes	Selected Causes: Communicable† Diseases	Selected Causes: Diseases of Respiratory System‡	All Other Causes
Frequency Rate (Disabling Illnesses per 1,000 Children)				
Total urban, all areas§	224.6	105.4	74.2	45.0
100,000 and over	211.7	98.4	73.9	44.4
25,000-100,000	220.0	103.8	71.9	44.2
Under 25,000	301.9	173.9	79.1	48.9
Frequency Index (Frequency Rate, Cities of 100,000 Population and Over = 100)				
Northeast				
25,000-100,000	95	109	76	100
Under 25,000	119	148	98	102
Central				
25,000-100,000	115	118	112	114
Under 25,000	181	247	105	121
West				
25,000-100,000	96	111	75	99
Under 25,000	122	148	99	102
South				
25,000-100,000	120	137	118	97
Under 25,000	112	148	93	94

* Illness disabling for seven consecutive days or longer in a twelve month period. All fatal and hospital cases are included without reference to the duration of disability.
† For the diseases included, see the second footnote, table 1.
‡ For the diseases included, see the third footnote, table 1.
§ Exclusive of cases and persons in families with income unknown.

cities than among children in relief families in cities with population of 100,000 and over; among children in families with income in excess of $3,000 the excess amounted to 81 per cent, and in the intermediate income classes the excess ranged from 85 to 96 per cent. On the other hand, in no income class was an excess of this order observed in the small-city rates for other causes of illness.

Important Specific Diseases of Children.—In the group of communicable diseases, the most frequently reported disabling illnesses of children under 15 years of age were measles, chickenpox, whooping cough, mumps and scarlet fever. In general, measles showed the highest incidence and diphtheria the lowest, the relative frequency of the other communicable diseases of childhood showing some variation in the

large, intermediate and small cities. These rates may be observed in table 4.

On the average, measles shows a higher frequency than the other communicable diseases of childhood, but the marked excess observed in this survey reflects an unusually high incidence of this disease in 1935, the approximate year of survey.[9] The relatively high incidence of mumps, especially in the two groups of cities with population under 100,000, is also notable.

TABLE 3.—*Frequency Rate and Frequency Index of Disabling Illness* According to Income and Sole or Primary Cause Among 518,767 White Children Under Fifteen Years of Age in Eighty-Three Cities Classified by Size in a Twelve Month Period*

Population Class of City and Income	All Causes	Selected Causes: Communicable† Diseases	Selected Causes: Diseases of Respiratory System‡	All Other Causes
Frequency Rate (Disabling Illnesses per 1,000 Children)				
Total urban, all incomes§	224.6	105.4	74.2	45.0
100,000 and over	211.7	93.4	73.9	44.4
25,000-100,000	220.0	103.8	71.9	44.2
Under 25,000	301.9	173.9	79.1	48.9
Frequency Index (Frequency Rate, Cities of 100,000 Population and Over = 100)				
Relief				
25,000-100,000	99	104	93	99
Under 25,000	136	182	101	102
Nonrelief				
Under $1,000				
25,000-100,000	102	109	97	99
Under 25,000	141	187	104	109
$1,000-$2,000				
25,000-100,000	108	120	94	101
Under 25,000	152	196	114	116
$2,000-$3,000				
25,000-100,000	111	111	117	98
Under 25,000	147	185	115	116
$3,000 and over				
25,000-100,000	113	113	121	95
Under 25,000	146	181	119	119

* See the first footnote, table 2.
† For the diseases included, see the second footnote, table 1.
‡ For the diseases included, see the third footnote, table 1.
§ See the fourth footnote, table 2.

In the preceding section, attention was directed to the high incidence of the group of communicable diseases among children in the small surveyed cities. The rates shown in table 4 indicate that the high frequency of measles and mumps among children in the small cities accounts largely for this excess. A higher incidence of measles in small, as compared with large, cities was observed also in a special analysis of cases of the notifiable diseases reported by health officers

9. Olesen, R., and Hampton, B. C.: Seasonal Patterns and Trends of Communicable Diseases, Pub. Health Rep. **52**:609, 1937.

TABLE 4.—*Frequency Rates of Important Disabling Diseases* According to Cause (Sole, Primary and Contributory) in a Sample of 518,767 White Children Under Fifteen Years of Age in Eighty-Three Cities Classified by Size; Rates by Specific Ages Among 373,446 White Children in Thirty-One Cities of 100,000 Population and Over in a Twelve Month Period*

Diagnosis	Population Class			Cities of 100,000 and Over Age Period, Yr.			
	100,000 and Over	25,000-100,000	Under 25,000	Under 1	1-4	5-9	10-14
	Frequency Rate per 1,000 Children Under 15 Years			Frequency Rate			
				Per 1,000 Live Births	Per 1,000 Children		
Communicable diseases							
Measles†	37.4	32.8	67.5	3.6	48.4	61.4	14.2
Chickenpox	18.8	15.5	27.2	8.1	27.8	29.9	4.8
Whooping cough	13.9	8.0	21.6	6.3	27.1	20.8	0.7
Mumps	11.5	32.5	47.4	0.9	8.1	19.0	8.4
Scarlet fever	11.1	9.5	11.7	...	10.2	17.1	7.9
Diphtheria	1.1	2.0	1.5	...	1.1	1.7	0.8
Acute diseases of the respiratory system							
Tonsillectomy, adenoidectomy	18.4	15.3	17.8	...	14.2	27.9	15.1
Influenza, grip	14.4	25.5	22.8	3.6	11.5	18.2	14.2
Colds	13.6	6.5	12.6	13.5	18.5	16.1	8.4
Tonsillitis	8.2	6.8	9.4	1.8	8.8	10.7	6.5
Pneumonia	7.9	11.0	9.6	13.5	13.8	8.5	2.9
Bronchitis	6.9	3.8	5.8	10.8	9.7	8.2	3.5
Chronic diseases and impairments							
Diseases of the nervous system	3.0	3.0	2.9	3.6	3.2	2.8	3.0
Diseases of the heart	2.1	1.0	1.8	8.1	0.5	2.2	2.2
Rheumatism	1.9	1.8	2.0	...	0.7	2.3	2.5
Nephritis	1.6	2.0	2.3	...	1.1	2.0	1.8
Orthopedic impairments	1.6	0.8	0.9	0.9	1.8	1.5	1.7
Acute diseases of the digestive system							
Appendicitis	3.9	4.0	3.8	...	1.4	3.7	6.2
Indigestion	1.7	2.0	3.2	...	2.9	1.5	1.2
Diarrhea, enteritis	1.0	1.8	2.3	5.4	2.0	0.5	0.3
All other diseases							
Ear and mastoid diseases	8.5	4.3	7.6	8.1	14.5	0.9	3.7
Cervical adenitis, other diseases of lymphatic system	2.4	1.5	1.8	...	4.5	2.6	1.4
Congenital malformations,‡ diseases of early infancy	1.3	1.5	0.9	19.9	0.2	...	0.3
Circumcision	0.7	1.0	1.2	2.7	1.1	0.8	0.1
Accidents							
Total§	11.2	10.5	14.3	1.9	10.2	11.6	12.8
Home	5.4	‖	‖	1.1	7.5	5.9	4.3
Other public	4.8	‖	‖	0.3	1.2	5.4	7.1
Automobile	1.9	‖	‖	0.3	1.1	2.2	2.3

* See the first footnote, table 2.

† Including German measles.

‡ Except congenital malformation of the heart, which is included with diseases of the heart.

§ Rates for accidental injuries by place of occurrence are based on the experience of 65,136 children under 15 years of age in eight cities of 100,000 population and over. For the cities included, see the third footnote, appendix to table 1. Rates are exclusive of contributory illnesses.

‖ Data not available.

to the United States Public Health Service in 1935;[10] the same tendency was apparent in reported cases of chickenpox.

The age trend of the frequency rates of disabling illness due to the communicable diseases was definitely upward during the early years of childhood, approaching a maximum in the age period 5 to 9 years and declining to a definitely lower level among children 10 to 14 years old. Whooping cough represents an exception to this trend, showing its maximum incidence in children of the preschool ages (1 to 4 years). Chart 2 shows the frequency rates of illness due to the communicable diseases at specific ages of childhood; the rates on which these curves are based are included in table 4. It should be noted again that the age variation in the incidence of illness considered here is characteristic of white urban children; the nature of the age trend differs in rural and in Negro children.

In the group of acute diseases of the respiratory system, the leading causes of disabling illness among children under 15 years of age were tonsillitis, influenza, colds, pneumonia and bronchitis; tonsillitis (including other pathologic conditions of the tonsils and adenoids which preceded operation) was the most frequently reported of these diseases. The disabling illness rate for pneumonia was approximately of the order of the rate for scarlet fever and several times higher than the rate for diphtheria (table 4).

Table 4 and chart 2 show the nature of the variation in the frequency rates of illness due to the diseases of the respiratory tract at specific ages of childhood. Tonsillitis (including tonsillectomies) and influenza occurred with greatest frequency in the age period 5 to 9 years. Pneumonia and bronchitis, however, showed a higher incidence among preschool children (ages 1 to 4 years) than among those aged 5 to 9.

Among other diseases showing a higher frequency among preschool children than among those aged 5 to 9 years, the diseases of the ear

10. The incidence rates of measles in 1935 were as follows (rates per thousand persons, all ages): in cities with population of 100,000 and over, East 5.4, Central 9.7, West 3.7, South 1.7; in cities of 10,000 to 25,000, East 11.3, Central 18.3, West 14.3, South, 4.0. The cases represent those reported by health officers to the United States Public Health Service; the cities selected include all reporting cities in the population classes specified above which were located in the eighteen states included in the National Health Survey. Data for cities with population under 10,000 were not available. The population used as a base for the rates is that of the 1930 Federal Census. The minimum number of cities reporting and the aggregate population were as follows: (1) Cities of 100,000 and over, East twenty-three cities and 13,867,534 persons, Central seventeen cities and 9,993,116 persons, West ten cities and 2,351,682 persons, South nine cities and 1,892,920 persons; (2) cities of 10,000 to 25,000, East eighty-three cities and 1,398,549 persons, Central twenty-eight cities and 493,627 persons, West eight cities and 130,020 persons, South seven cities and 118,628 persons.

and mastoid process may be noted. Accidental injuries occurring in the home likewise were most frequent among children of the preschool ages. In the succeeding years of childhood, the frequency of accidents in the home declined but public accidents showed an upward trend.

Among infants born during the survey year, the acute diseases of the respiratory system considered as a group represented the major cause of disabling illness. As a cause of illness, this group of diseases outranked in frequency congenital malformations and other conditions commonly designated as the diseases of early infancy which are the lead-

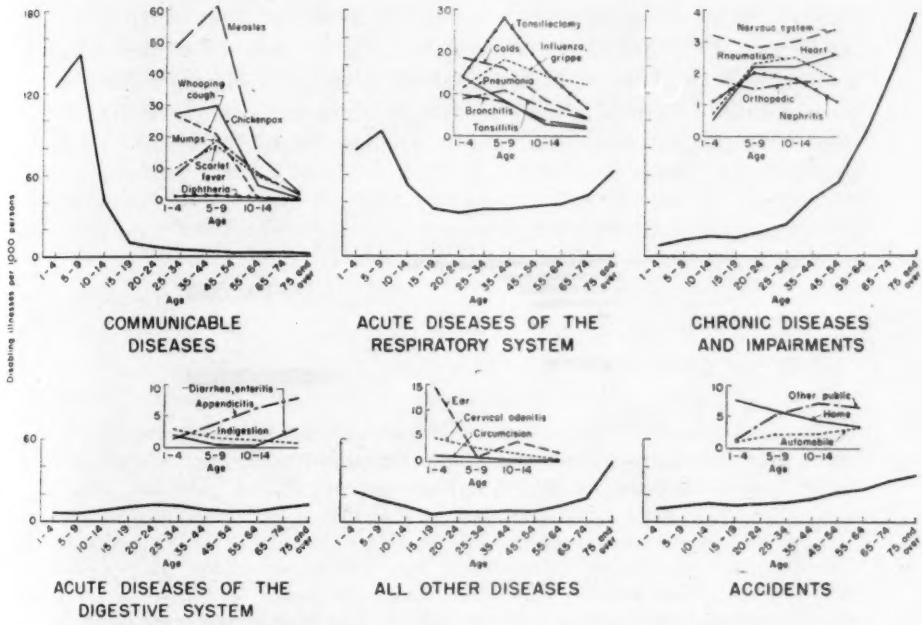

Chart 2.—Age variation in frequency rates of disabling illness in a twelve month period, classified by cause in broad disease groups (sole or primary causes only) and, for the period of childhood, by specific causes (sole, primary and contributory causes). Conditions associated with the puerperal state and diseases of the female genital organs are not included. This represents a sample of 1,581,577 white persons in thirty-one cities with population of 100,000 and over (exclusive of persons in families with income unknown) canvassed in 1935-1936. The rates for infants under 1 year of age are not shown, since the period of exposure was less than twelve months (fourth column, table 4).

ing causes of infant mortality. The acute diseases of the respiratory tract were more frequent causes of illness among infants than the acute communicable diseases; among children of preschool age (1 to 4 years) and early school age (5 to 9 years), the relative importance of these two groups of diseases was reversed.

PREVALENCE OF PERMANENT ORTHOPEDIC IMPAIRMENTS

The illnesses of children considered in the preceding section were those which caused disability lasting for seven consecutive days or longer in the survey year. It has been shown (table 1) that 12 in every 10,000 surveyed white children under 15 years of age had been disabled for a week or longer in the twelve month period by permanent orthopedic impairments. The severity of these disablements is indicated by their mean duration, which was almost eight months.

The survey included also an enumeration of cases of permanent orthopedic impairment which had caused no incapacity in the survey year and cases of impairment resulting in disability of less than seven consecutive days' duration. The combination of cases of nondisabling and cases of disabling impairment of all durations makes possible the computation of the total prevalence rate of permanent orthopedic impairments. This total prevalence rate in children under 15 years of age was 49.5 per 10,000.

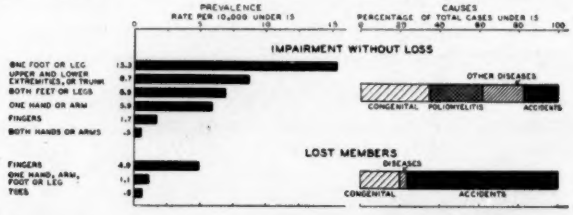

Chart 3.—Prevalence rate of disabling and nondisabling orthopedic impairments (sole, primary and contributory causes) classified by the part affected, and the percentage distribution of impairments by cause, for 602,814 white and colored children in eighty-three cities canvassed in 1935-1936. The prevalence rates of impairments not shown here are as follows (rates per 10,000 persons under 15 years of age): Impairment without loss: toes, 0.3; trunk (except joints, spine, back, side), 1.7; lost members: upper and lower extremities, both hands or arms, 0.1; other impairments (with or without loss), the part involved not being reported, 1.8. In the distribution of impairments by cause, the last group was included with impairments without loss of member.

Chart 3 shows graphically the prevalence rate of disabling and nondisabling orthopedic impairments, classified according to the nature of the impairment and the part affected, in children under 15 years of age; only rates of 0.5 per 10,000 or higher are shown here. It will be observed that with the exception of lost fingers, impairments involving loss of members were relatively infrequent. On the other hand, the prevalence rates of crippling impairments without loss of the member which involved one or both lower extremities, upper and lower extremities in combination or the trunk were relatively high.

Chart 3 also shows the percentage distribution of orthopedic impairments among children under 15 years of age by cause. In the age

group under 15 years, congenital defects, poliomyelitis and accidental injuries accounted for 79 per cent of all impairments which did not involve loss of the member, and accidents alone accounted for 77 per cent of all losses of members.

MEDICAL AND NURSING CARE OF DISABLING ILLNESS DUE TO ALL CAUSES

Age Variation in the Receipt of Medical Care.—The illnesses of childhood, except in the period of infancy, are characterized by a shorter mean duration and a higher recovery rate than those of adult life. It may be expected, therefore, that children and adults will differ in respect to the proportion of illnesses receiving medical and nursing care and in respect to the intensity of care per patient attended. The experience of white persons in eighty-three cities canvassed in the survey is analyzed from this standpoint in table 5. Since the number of physicians and nurses and the facilities of hospitals and their associated outpatient departments tend to vary with the degree of urbanization, the data are presented for the surveyed population in three groups of cities classified by size. Chart 4 graphically shows the results for the large and small cities only.

Columns 1 and 5 of the table relate to medical attendance of disabling illness by a physician in the home, clinic or physician's office but exclude cases in which medical care was received only in the hospital. Furthermore, cases in which care was given by a physician in the home, office or clinic in addition to hospital care are counted both as physician's and as hospital cases, but the consultations with a physician in such cases exclude those received during hospitalization. Columns 2 and 6, designated as "Hospital, General," represent an approximation of general hospital patients and patient days obtained by excluding all cases of tuberculosis treated in a hospital and cases of patients with nervous and mental disease or defect in which the patient had received institutional care for at least a year; it was assumed that in the majority of such cases hospitalization was in special institutions.[11]

Reference to table 5 indicates that the proportion of the disabling illnesses of children under 15 years of age receiving care from a physician was, in general, lower than among adults in each group of surveyed

11. Cases of patients newly admitted to hospitals for the mentally diseased during the survey year could not be segregated on the basis of data recorded in the survey, and these cases are therefore included in the general hospital group. In the interpretation of the data relating to hospital care, it should be noted that the survey enumerators sought information concerning all cases in which hospital care was received, without restriction as to the period of disability; it is believed, however, that cases of hospitalized patients disabled for less than a week were incompletely enumerated.

cities; however, the age variation was not marked except in the small cities (population under 25,000). The proportion of illnesses receiving general hospital care was approximately the same in childhood as in old age but was lower in both of these periods than in the age period

TABLE 5.—*Age Variation in the Receipt of Medical and Nursing Care of Disabling Illness* Among 2,152,740 White Persons† in Eighty-Three Cities of Three Population Classes in a Twelve Month Period*

Age Period, Yr.	Percentage of Disabling Illnesses* Receiving Specified Care				Services per Case of Disabling Illness* Receiving Specified Care				Number of Disabling Illnesses*
	Medical		Bedside Nursing		Medical		Bedside Nursing		
	Physician	Hospital, General‡	Private Duty Nurse§	Visiting Nurse	Physician	Hospital, General‡	Private Duty Nurse§	Visiting Nurse	
Cities of 100,000 and Over									
All ages†.....	71.0	30.2	3.4	7.7	7.5	19.2	28.7	5.2	253,581
Under 15....	68.1	19.5	1.2	13.9	4.3	15.3	18.7	3.2	79,053
15-24.....	68.5	44.1	2.9	7.0	6.4	15.2	13.3	5.8	32,519
25-64.....	72.9	35.7‖	4.3	4.6	9.0	20.6‖	23.9	8.0	118,745
65 and over.	74.9	19.7	6.7	3.0	11.0	32.6	60.1	14.0	23,264
Cities of 25,000-100,000									
All ages†.....	72.1	23.1	4.8	4.7	7.4	16.2	21.2	6.5	55,990
Under 15....	63.9	12.5	1.7	6.2	4.6	11.6	10.8	3.8	17,252
15-24.....	73.7	32.8	4.5	5.1	6.3	12.6	11.9	5.1	7,925
25-64.....	76.6	29.2‖	6.3	3.8	8.7	17.4‖	18.3	8.0	25,223
65 and over.	74.9	14.7	7.8	3.6	10.5	28.2	46.2	15.7	5,590
Cities Under 25,000									
All ages†.....	68.7	18.9	4.4	6.0	7.1	16.7	23.3	5.2	59,431
Under 15....	57.6	8.8	1.4	9.2	4.3	11.1	11.3	2.9	20,198
15-24.....	70.5	27.7	4.7	6.0	6.1	12.5	9.5	4.9	8,744
25-64.....	75.3	25.7‖	5.9	4.0	8.5	18.5‖	20.1	8.2	24,374
65 and over.	75.9	13.1	8.1	3.2	10.2	28.4	50.5	12.2	6,120

* Illness disabling for seven consecutive days or longer in a twelve month period. All confinements and fatal and hospital illnesses are included without reference to the duration of disability. Illness as used here is a continuous period of disability, whether due to a single cause or multiple causes.

† Exclusive of persons of unknown age or unknown income.

‡ Cases of patients receiving hospital care are exclusive of all cases of hospitalized patients with tuberculosis, and cases of patients with nervous and mental disease or defect remaining in institutions for twelve months. These exclusions leave a group of hospital cases which roughly approximate patients treated in general and special hospitals, exclusive of hospitals for the tuberculous and the mentally diseased. Since the type of institution in which care was received was not recorded in the survey, a more exact definition of general hospital patients is not possible.

§ Includes attendance in home or hospital.

‖ Excluding also hospitalized confinements terminating in live births, the proportion of disabling illnesses hospitalized was as follows: cities of 100,000 and over, ages 15-24, 35.7 per cent; ages 25-64, 30.7 per cent; cities of 25,000 to 100,000, ages 15-24, 28.0 per cent; ages 25-64, 25.9 per cent; cities under 25,000, ages 15-24, 24.7 per cent; ages 25-64, 23.2 per cent. The corresponding average numbers of hospital patient days were as follows: 17.9 (ages 15-24) and 23.7 (ages 25-64) in cities of the first class; 14.9 (ages 15-24) and 19.6 (ages 25-64) in cities of the second class; 13.8 (ages 15-24) and 20.4 (ages 25-64) in cities of the third class.

between 15 and 65 years. This period of adult life includes the childbearing ages, in which hospital care of women for conditions associated with the puerperal state is frequent. At each age period, the proportion of hospitalized illnesses was lower in the cities of population under 25,000 than in the large cities, but the difference was most marked among children and the aged.

In each group of cities, the illnesses of children receiving medical care were given less intensive care than the attended illnesses of adults. Among children under 15 years of age, in the average case of attended disabling illness the patient had approximately 4 consultations with a physician, exclusive of hospital visits; in youth, the average was 6 consultations, in the ages between 25 and 65, approximately 9, and in old age, between 10 and 11.

In the large cities the average length of stay in the hospital per patient among children was 15.3 days; among adults 25 to 64 years of age, 20.6 days, and among the aged, 32.6 days; in youth the average number of hospital patient days was approximately the same as in childhood. In the cities with population under 25,000, the average duration

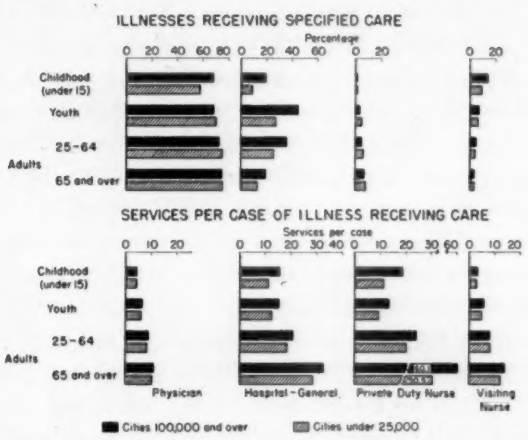

Chart 4.—Percentage of disabling illnesses occurring in a twelve month period which received medical and nursing care, and services per case of illness receiving care, by age, for 1,581,577 persons in thirty-one cities with population of 100,000 and over and for 267,953 persons in forty-two cities with population under 25,000 canvassed in 1935-1936. Cases of illness receiving hospital care are exclusive of all hospital cases of tuberculosis and cases of mental and nervous disease treated in institutions for twelve months.

of hospitalization was somewhat lower at each age period than in the large cities, but the relative variation by age was of the same nature. In general, the average length of stay in the hospital observed in the survey exceeds the average for general hospitals as usually reported. The lack of agreement arises from the fact that the surveyed persons designated as general hospital patients include the residual group of the mentally diseased admitted to special institutions during the survey year and exclude certain hospital patients disabled for less than a week, who were incompletely enumerated.

The low average intensity of medical care given to children results from the fact that the majority of child patients are treated for diseases with low severity and a favorable prognosis. As age increases the severity of illness increases, and medical care is required in correspondingly greater amount.

In comparison with medically attended illnesses, the proportion of illnesses receiving bedside nursing care was relatively low at each age period. The visiting nurse was used more frequently than the private duty nurse for bedside nursing care of the illnesses of children; with increasing age, there were, in general, an increase in the proportion of illnesses cared for by a private duty nurse and a decrease in the proportion attended by a visiting nurse. The amount of bedside nursing care received, measured as days of care by the private duty nurse or as visits of the visiting nurse, was, in general, lower among children than among adults, exclusive of the period of youth.

Income and Medical Care of Childhood Illness.—While the illnesses of children received, on the average, less medical care than those of adults, measured in terms of both the proportion of illnesses treated and the amount of care per patient, it was found that the experience of children in families at different income levels showed a wide departure from the average. This relation may be examined in table 6, in which the results are again presented separately for children in three groups of surveyed cities classified by size.

Care was given by a physician (exclusive of hospital medical care) in 80 per cent of the cases of disabling illness of children in families with an income of $3,000 and over in the large surveyed cities; in relief families in these cities in only 65 per cent of the cases was medical care received at home or in the office. With decreasing urbanization, represented by the intermediate and small surveyed cities, the proportion of cases in which children received care from a physician was lower than in the large cities at each income level, but the relative difference between the lowest and the highest income group was of the same order as in the large cities.[12] A similar association between income and the intensity of care received from a physician was observed. In families with an income of $3,000 and over, each child patient received, on the average, about 5 consultations with a physician in the home, clinic or physician's office; in families on relief the average was about 4 consultations.

12. In the rural areas of sixteen counties canvassed in Georgia, the proportion of the disabling illnesses of Negro children receiving care from a physician was notably low. Only 39 per cent of 680 disabling illnesses recorded among Negro children under 15 years of age received care from a physician, exclusive of hospital medical care; among white children, the comparable figure was 64 per cent (1,479 disabling illnesses being recorded). The results of the survey of children in rural areas will form the subject of a separate report.

Further examination of the data in table 6 indicates that the proportion of children's illnesses attended by a physician (exclusive of hospital treatment) and the intensity of such care were approximately the same in self-sustaining families with incomes below $1,000 as in families on relief; in the group at the next income level (between $1,000 and

TABLE 6.—*Variation in Receipt of Medical and Nursing Care of Disabling Illness* According to Income Among 518,767 White Children† Under Fifteen Years of Age in Eighty-Three Cities of Three Population Classes in a Twelve Month Period*

Income Class	Percentage of Disabling Illnesses* Receiving Specified Care				Services per Case of Disabling Illness* Receiving Specified Care				Percentage Distribution of Disabling Illnesses* by Income
	Medical		Bedside Nursing		Medical		Bedside Nursing		
	Physician	Hospital, General‡	Private Duty Nurse§	Visiting Nurse	Physician	Hospital, General‡	Private Duty Nurse§	Visiting Nurse	
Cities of 100,000 and Over									
All incomes†.....	68.1	19.5	1.2	13.9	4.3	15.3	18.7	3.2	100.0
Relief.....	65.1	24.3	0.4	19.5	3.9	19.6	20.2	3.7	27.2
Nonrelief									
Under $1,000...	63.0	19.6	0.6	13.5	4.3	15.9	15.2	3.2	15.8
$1,000-$2,000....	68.4	17.5	1.1	12.3	4.3	13.4	19.3	2.7	39.1
$2,000-$3,000....	74.5	16.7	2.1	10.5	4.6	9.5	17.9	2.8	11.7
$3,000 and over	80.2	16.1	5.8	7.7	5.4	9.8	19.0	2.5	6.3
Cities of 25,000-100,000									
All incomes†.....	68.9	12.5	1.7	6.2	4.6	11.6	10.8	3.8	100.0
Relief.....	60.9	14.9	0.4	10.3	4.1	14.0	5.8	3.6	26.1
Nonrelief									
Under $1,000...	60.3	10.9	0.8	5.4	4.4	12.1	10.4	4.5	21.7
$1,000-$2,000....	64.2	11.4	1.6	5.0	5.0	10.3	11.1	4.1	36.9
$2,000-$3,000....	71.2	12.9	3.1	3.3	4.7	9.8	12.5	2.0	9.7
$3,000 and over	76.7	13.3	8.9	1.8	5.5	7.9	10.8	4.2	5.6
Cities Under 25,000									
All incomes†.....	57.6	8.8	1.4	9.2	4.3	11.1	11.3	2.9	100.0
Relief.....	52.6	7.8	0.5	13.4	3.9	17.1	6.0	3.1	25.6
Nonrelief									
Under $1,000...	54.0	8.2	1.0	9.0	4.3	12.3	10.4	2.8	24.6
$1,000-$2,000....	59.7	9.2	1.4	8.3	4.4	8.7	9.8	2.8	36.3
$2,000-$3,000....	66.5	10.4	3.2	4.5	4.7	7.5	14.6	2.6	9.3
$3,000 and over	71.6	11.8	3.6	4.6	5.1	4.5	15.7	1.4	4.2

* See the first footnote, table 5.
† Exclusive of children of these ages in families for which income was reported as unknown.
‡ See the third footnote, table 5.
§ See the fourth footnote, table 5.

$2,000), the amount of care did not greatly exceed that in the two groups with the lowest incomes. In this survey, the proportion of all disabling illnesses of children under 15 years of age occurring in families below the $2,000 income level ranged from 82 per cent in the large cities to 87 per cent in the cities with population under 25,000. Thus, the majority of childhood illnesses occurred in the economic groups in which the proportion of illnesses receiving care from a physician and the amount of care per case were relatively low. In this connection, it should be recalled that the illnesses of children considered here had

caused disability of at least a week's duration, and medical supervision may be assumed to be necessary in the majority of such cases.

In the small surveyed cities, the proportion of the illnesses of children receiving medical care in the hospital was lowest in relief families and increased progressively as family income increased. In the large cities, however, this association was reversed, the proportion of cases in which children were hospitalized being highest in families on relief and decreasing as family income increased. This situation reflects the greater demand for hospital care resulting from the congested housing conditions of families with low incomes in the large cities and the more abundant supply of free hospital facilities by which this demand can be met. The relation will be further clarified in the discussion of the data shown in table 7.

The association between family income and the type of nurse employed for bedside nursing care of the sick, i. e., private duty or visiting nurse, reflects the method of meeting the costs of these services. The bedside nursing care given by visiting nurses is provided largely without cost to the patient, while the costs of private duty care are met from private income. It is consistent with these facts that a relatively high proportion of children's illnesses in families with low incomes received care from a visiting nurse, while in these families the proportion of children's illnesses which were cared for by a private duty nurse was negligible. The essential question presents itself: Does the bedside nursing care provided by visiting nurses for the illnesses of children in families with low incomes meet their needs as adequately as the care given by private duty nurses? The question is raised since such a consideration should underlie the interpretation of the relation observed between economic status and the type of bedside nursing care received. Data bearing on the question are, however, beyond the scope of the present survey.

Additional factors of importance in an analysis of the association between economic status and hospital care are considered in table 7, in which the hospital cases of children are classified in two broad groups according to the type of treatment received, i. e., medical and surgical. The figures in this table indicate that the proportion of children who became hospital patients during the survey year (as distinguished from the proportion of illnesses which were treated in a hospital) was notably higher in the large cities than in the intermediate and small cities. This excess is not the result of a higher incidence of illness (since the frequency rate of illness was found to be lowest in the large cities) but is due, in part, to a relatively higher frequency of medical hospital cases. Among children under 15 years of age, the frequency rate of medical hospital cases in the large cities was 15 per thousand and in the small

cities about 7 per thousand. On the other hand, the frequency of surgical hospital cases showed relatively smaller variation, the frequency rate in the large cities being 27 per thousand children under 15 years and in the small cities 20 per thousand.

TABLE 7.—*Frequency Rate of Hospital Cases* Classified as Medical and Surgical and of Nonhospital Surgical Cases, According to Income Among 518,767 White Children† Under Fifteen Years of Age in Eighty-Three Cities of Three Population Classes in a Twelve Month Period*

Income Class	Cases per 1,000 Children				
	Hospital			Surgical, Patient Treated in Home, Clinic or Physician's Office‡	Surgical, Total Hospital and Nonhospital
	Total	Medical	Surgical		
Cities of 100,000 and Over					
All incomes†	41.7	14.9	26.7	10.0	36.7
Relief	50.5	26.9	32.6	7.9	40.5
Nonrelief					
Under $1,000	37.8	14.9	23.9	9.4	32.3
$1,000-$2,000	34.8	10.7	24.1	10.2	34.3
$2,000-$3,000	36.9	8.8	28.1	13.5	41.5
$3,000 and over	39.0	7.6	31.4	11.6	42.9
Cities of 25,000-100,000					
All incomes†	27.8	8.7	19.1	12.7	31.7
Relief	36.2	14.1	22.1	9.7	31.8
Nonrelief					
Under $1,000	21.6	7.7	13.9	10.9	24.8
$1,000-$2,000	24.5	6.1	18.4	14.2	32.6
$2,000-$3,000	31.6	6.5	25.1	14.7	39.9
$3,000 and over	36.2	10.8	25.5	20.7	46.1
Cities Under 25,000					
All incomes†	26.9	6.5	20.4	12.4	32.8
Relief	26.2	7.6	18.6	13.1	31.7
Nonrelief					
Under $1,000	22.3	3.3	17.0	9.4	26.5
$1,000-$2,000	27.8	6.6	21.2	12.5	33.7
$2,000-$3,000	33.3	5.5	27.9	17.4	45.3
$3,000 and over	41.5	10.3	31.2	17.7	48.9

* Including cases in a twelve month period (1) in which the hospitalized illness or injury was the sole cause of disability and (2) in which multiple causes were assigned to the illness and hospital care was received for the primary, or any contributory, cause of the illness. In enumerating hospital cases, no limitation was imposed concerning the duration of the disability.

† Exclusive of children of these ages in families for which income was reported as unknown.

‡ Including only illness disabling for seven consecutive days or longer which received surgical treatment as specified.

It is notable, furthermore, that the frequency rate of medical hospital cases in the large cities decreased consistently with rise in income, while the frequency of surgical hospital cases increased with increasing income, if the rate for children in relief families is excepted. The association between income and the frequency of surgical cases in which treatment was given outside the hospital was likewise direct, the lowest rate being observed in the group with the lowest incomes, with a progressive increase in succeeding income classes.

It appears, then, that in the large cities families with low incomes tend to hospitalize children in certain medical cases of illness which families in the higher income groups care for at home. The higher frequency rates of these cases in the low income groups do not imply that care is more adequate among the poor but indicate only that in a higher proportion of medical cases treatment is given outside the home.

MEDICAL AND NURSING CARE OF THE DISEASES OF CHILDREN CLASSIFIED BY CAUSE

Diseases of Children Treated by the Physician and Nurse.—The diseases of children which predominate among the child patients of the physician and nurse may be illustrated by the records of children in the large surveyed cities, shown in table 8; the experience of children in the intermediate and small cities shows no essential differences and is therefore omitted.

The combined frequency rate of disabling illness due to the communicable diseases, tonsillitis and other minor diseases of the respiratory tract among surveyed children in these large cities represented 75 per cent of the rate for all causes of illness in a twelve month period. It is thus consistent with the high incidence of these diseases that they were most frequent among children's illnesses treated by a physician, accounting for 71 per cent of the physicians' cases. Illnesses due to these causes, however, absorbed only 54 per cent of the physicians' services for children. On the other hand, the less frequent cases of pneumonia, the major chronic diseases, orthopedic impairments and accidental injury together represented only 15 per cent of the physicians' child patients but absorbed 28 per cent of their services for children. Illnesses due to causes included in the latter group are relatively severe and require intensive medical supervision.

The communicable diseases and minor diseases of the respiratory tract occurred in about the same proportion among children receiving bedside nursing care from a private duty nurse in the home as among those attended by a physician. Cases of pneumonia accounted for a relatively large proportion of the private duty nurses' child patients and for 18 per cent of the total days of private duty nursing care received by children in the home or hospital.

The communicable diseases and minor diseases of the respiratory tract accounted for 86 per cent of the child patients of visiting nurses and for 74 per cent of their visits to children. Only 2.6 per cent of all cases of children attended by a visiting nurse were included under major chronic diseases and orthopedic impairments, and these cases represented only 6.3 per cent of the nurses' visits. Pneumonia accounted for 3 per cent of all cases of children attended by a visiting nurse and absorbed 8 per cent of all nursing visits to children.

Diseases of Children Receiving Hospital Care.—The frequency rates of hospital cases among children under 15 years of age according to cause are shown in chart 5, which includes rates for surveyed children

TABLE 8.—*Frequency Rate of Disabling Illness* Due to Selected Causes (Sole or Primary Only) Receiving Treatment from a Physician, Private Duty Nurse or Visiting Nurse and Rate of Physicians' and Nurses' Services Among 373,446 White Children Under Fifteen Years of Age in Thirty-One Cities of 100,000 Population and Over in a Twelve Month Period*

Diagnosis	Disabling Illnesses* Receiving Specified Care per 1,000 Children				Services for Disabling Illness* per 1,000 Children			Frequency Rate†† of Disabling Illness*
	Physician, Home, Office or Clinic	Private Duty Nurse		Visiting Nurse	Physician, Home, Office or Clinic	Private Duty Nurse,† Home and Hospital	Visiting Nurse	
		Hospital	Home					
All causes	144.2	1.07	1.53	29.53	623.9	48.61	93.71	211.7
Selected causes								
Communicable diseases‡	58.4	0.11	0.69	21.41	203.6	15.64	57.43	93.4
Diseases of respiratory system								
Total	51.0	0.36	0.66	5.01	192.2	15.21	19.56	73.9
Tonsillitis	20.0	0.17	0.10	1.65	50.3	0.87	4.06	25.7
Other minor diseases§	24.4	0.04	0.24	2.39	82.8	5.73	7.91	40.7
Pneumonia	6.6	0.15	0.32	0.97	59.1	8.61	7.59	7.5
Diseases of digestive system								
Total	5.9	0.28	0.01	0.38	25.2	3.86	1.83	7.0
Appendicitis	3.4	0.25	‖	0.16	14.3	3.08	0.75	3.8
Other diseases¶	2.5	0.03	0.01	0.22	10.9	0.78	1.08	3.2
Major chronic diseases								
Total	5.8	0.05	0.06	0.63	50.4	3.40	4.50	7.9
Tuberculosis, all forms	0.3	....	0.01	0.06	2.8	0.10	0.50	0.5
Nervous and mental diseases#	1.7	0.03	0.01	0.18	12.2	0.86	0.96	2.7
Rheumatism	1.0	‖	0.01	0.11	10.2	1.22	0.98	1.2
Degenerative diseases**	2.8	0.02	0.04	0.28	25.2	1.22	2.06	3.5
Orthopedic impairments	0.8	0.02	‖	0.13	10.9	0.81	1.40	1.2
Accidents	8.8	0.07	0.02	0.40	56.8	5.07	1.67	10.8
All other causes	13.5	0.18	0.08	1.56	84.8	4.54	7.23	17.4

* See the first footnote, table 5.

† In this survey the days of care by a private duty nurse in the home and in the hospital were not recorded separately.

‡ Including chiefly the communicable diseases of childhood: measles, mumps, chickenpox, whooping cough, scarlet fever and diphtheria.

§ Including influenza, coryza, bronchitis, pleurisy, sinusitis, asthma, hay fever and other diseases of the respiratory system except tonsillitis, pneumonia and respiratory tuberculosis. In childhood the minor respiratory diseases predominate.

‖ The rate is 0.008 per 1,000, representing 1 illness of this category.

¶ Including indigestion, biliousness, diarrhea and enteritis, ulcer of the stomach or duodenum, diseases of the gallbladder or liver and other diseases of the digestive system except appendicitis.

Including mental defects.

** Including cancer; diabetes; cerebral hemorrhage and other forms of paralysis; diseases of the heart, arteriosclerosis and high blood pressure, and other diseases of the circulatory system, exclusive of hemorrhoids and varicose veins; nephritis and other nonvenereal diseases of the genitourinary system, exclusive of diseases of the female genital organs.

†† The rate is computed per 1,000 children.

in the large cities and in cities of population under 25,000. The hospital cases considered represent chiefly those in which treatment was given in general and special hospitals, exclusive of institutions for the care of the tuberculous and the mentally diseased.

In the surveyed cities with population of 100,000 and over, the total incidence of "general" hospital cases among children under 15 years of age was 41.2 per thousand. In the cities with population under 25,000, the rate was 26.6. These rates are equivalent to a ratio of 1 hospital patient for every 24 children under 15 years of age in the large cities and 1 patient for every 38 children in the small cities.

As a group, illnesses which were chiefly surgical [13] were most frequent among the hospitalized illnesses of children. Diseases of the tonsils and adenoids, appendicitis, diseases of the ear and mastoid process and accidents together accounted for 61 per cent of the hospital cases in the large cities and for 69 per cent in the small cities. Diseases of the tonsils and adenoids alone accounted for 37 per cent of all child hospital patients in the large cities and for 41 per cent of those in the small cities.

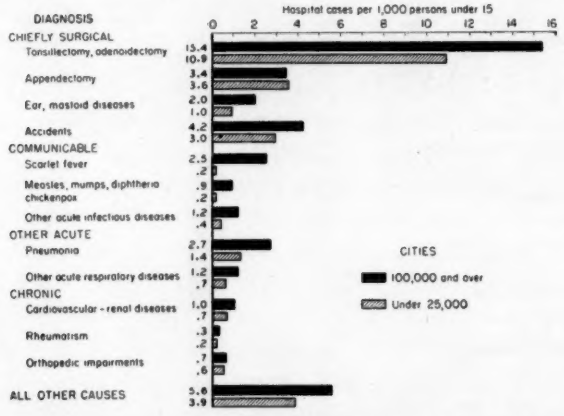

Chart 5.—Frequency rate of hospital cases classified by cause among children under 15 years of age in thirty-one cities with population of 100,000 and over and in forty-two cities with population under 25,000 canvassed in 1935-1936. "All other causes" is exclusive of all hospital cases of tuberculosis and of cases of mental and nervous disease or defect treated in an institution for twelve months.

Practice in regard to hospitalization for the acute communicable diseases of children showed characteristic differences with degree of urbanization, the frequency of hospital cases being relatively higher in the large than in the small cities. Hospital cases of pneumonia were relatively frequent in both groups of cities.

13. In the cities with population of 100,000 and over, tonsillectomies were performed in 98 per cent of the hospital cases of tonsillitis and appendectomies in 94 per cent of the cases of appendicitis; surgical treatment was given in 84 per cent of the cases of ear and mastoid disease and in 68 per cent of the accident cases. In the cities with population under 25,000 the corresponding figures were as follows: tonsillitis 99 per cent, appendicitis 97 per cent, diseases of the ear and mastoid 84 per cent and accidents 75 per cent.

Medical Care of Childhood Illness Classified by Cause and Income.—In a preceding section, the association between family income and medical care of childhood illness was examined, treatment received from a physician in the home, office or clinic and hospital medical care being considered separately. These groups are not mutually exclusive, since in certain cases patients treated in the hospital were also cared for by a physician before or after hospitalization. This duplication can be

TABLE 9.—*Percentage of Disabling Illnesses* Receiving No Medical Care According to Income and Sole or Primary Cause Among 518,767 White Children† in Eighty-Three Cities of Three Population Classes in a Twelve Month Period*

Diagnosis	Percentage of Disabling Illnesses Receiving No Medical Care					
		Relief Status and Annual Family Income				
			Nonrelief			
	All Incomes†	Relief	Under $1,000	$1,000-$2,000	$2,000-$3,000	$3,000 and Over
Cities of 100,000 and Over						
All causes	27	28	32	28	23	18
Selected causes						
Communicable diseases‡	37	37	43	38	31	22
Minor diseases of the respiratory system, except tonsillitis§	39	44	46	39	31	27
All other causes	9	10	11	9	7	6
Cities of 25,000-100,000						
All causes	34	36	38	34	27	21
Selected causes						
Communicable diseases‡	49	53	54	49	39	32
Minor diseases of the respiratory system, except tonsillitis§	39	45	46	37	29	22
All other causes	10	12	12	9	6	5
Cities Under 25,000						
All causes	41	46	45	39	33	26
Selected causes						
Communicable diseases‡	54	60	58	51	43	31
Minor diseases of the respiratory system, except tonsillitis§	43	47	48	40	36	33
All other causes	13	17	16	10	7	11

* See first footnote, table 5.
† Exclusive of children of these ages in families for which income was reported as unknown.
‡ For the diseases included, see the third footnote, table 8.
§ For the diseases included, see the fourth footnote, table 8.

eliminated by considering all illnesses receiving medical care from a physician, whether in the home, office, clinic or hospital. Such illnesses form the basis for the rates shown in table 9, the proportion of unattended illnesses representing cases in which no medical care was received from a physician in the home, office, clinic or hospital. The proportion of children's illnesses receiving no medical care in a twelve month period is shown for all causes of illness and for two major groups of children's diseases, according to relief status and family income. Chart 6 presents the results graphically, the percentage of unattended illnesses by cause

being shown only for children in a low income group (nonrelief, incomes less than $1,000) and in the class with incomes of $3,000 or over.

The results indicate that the largest proportion of unattended illnesses were included in the communicable group and the group of minor diseases of the respiratory tract, exclusive of tonsillitis. In the large cities, 37 per cent of all disabling illnesses due to the communicable diseases and 39 per cent of those due to minor diseases of the respiratory tract, exclusive of tonsillitis, received no medical care in the survey year, as compared with 9 per cent of illnesses due to all other causes. In the cities with population under 25,000 in 54 per cent of all cases of

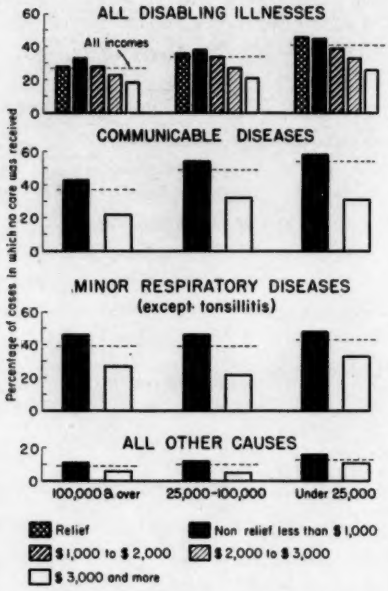

Chart 6.—Percentage of disabling illnesses occurring in a twelve month period which received no medical care, among surveyed children under 15 years of age classified by size of the city of residence, according to annual family income; and, for children in families with annual income under $1,000 (not on relief) and in families with annual income of $3,000 and over, the percentage of disabling illnesses due to certain broad groups of diseases which received no medical care. The data relate to eighty-three cities canvassed in 1935-1936.

communicable disease the patients were without medical attendance. The proportion of cases of unattended illness decreased with increasing family income, but at each income level in the highest proportion of such cases illnesses were due to the communicable diseases and to minor diseases of the respiratory tract, exclusive of tonsillitis.

The illnesses of children in families in the upper income groups which had been attended by a physician received more intensive care than the

attended illnesses in low income families, as is shown in table 10. In the average case a communicable disease among children in relief families received 3 visits, as compared with almost 5 visits in families with an income of $3,000 and over. For pneumonia, the average was approximately 7 visits per case for children in relief families and 12 visits per case for children in the highest income group; for tonsillitis, the corresponding figures were 2 as compared with 3 visits and for

TABLE 10.—*Physicians' Services Per Case of Disabling Illness* Attended for Selected Causes (Sole or Primary Only) According to Income Among 373,446 White Children† Under Fifteen Years of Age in Thirty-One Cities of 100,000 Population and Over in a Twelve Month Period*

Diagnosis‡	Services per Case Treated by Physician in Home, Office or Clinic					
			Nonrelief			
	All Incomes†	Relief	Under $1,000	$1,000-$2,000	$2,000-$3,000	$3,000 and Over
All causes	4.3	3.9	4.3	4.3	4.6	5.4
Selected causes						
Communicable diseases	3.5	3.0	3.3	3.4	4.1	4.8
Diseases of the respiratory system						
Tonsillitis	2.5	2.3	2.6	2.5	2.6	2.9
Other minor respiratory diseases	3.4	3.0	3.0	3.4	3.6	4.4
Pneumonia	8.9	6.8	8.5	10.1	10.8	12.4
Diseases of the digestive system						
Appendicitis	4.2	3.2	4.7	4.4	4.7	4.3
Other digestive diseases	4.4	4.0	4.4	4.6	4.6	5.5
Major chronic diseases						
Tuberculosis, all forms	10.0	§	§	§	§	§
Nervous and mental diseases	7.1	5.9	7.3	7.9	8.4	7.1‖
Rheumatism	10.1	7.1	12.3	9.2	14.1	19.4‖
Degenerative diseases	8.9	6.8	8.4	8.8	12.5	15.0
Orthopedic impairments	14.3	15.5	14.7	12.7	12.4‖	21.0‖
Accidents	6.5	6.3	6.9	6.3	6.2	7.8
All other causes	6.2	5.2	5.8	6.6	7.0	9.1

* See the first footnote, table 5.
† See the second footnote, table 9.
‡ For the definition of the diagnosis groups, see the footnotes to table 8.
§ The rate is shown for all incomes only because of the small number of cases.
‖ The rate represents the experience of 25 cases of treated illness or less.

other minor respiratory diseases 3 as compared with 4 visits. The same tendency toward more intensive care in the families with higher incomes was observed for other diseases of childhood. The nature of the income differential was the same in cities of each population class, and figures for the intermediate and small cities are therefore not shown here.

SUMMARY

In a canvass of eighty-three representative urban communities conducted by the United States Public Health Service in 1935-1936, records of illness and medical and nursing care received in a twelve month

period were obtained for 518,767 white children under 15 years of age. The records of sickness were limited to disabling illnesses, i. e., those which had prevented the usual activities of the preschool child or school attendance of the school child for at least seven consecutive days in the twelve month period. The characteristics of the disabling diseases of childhood and their medical and nursing care as observed in the survey may be summarized as follows:

Disabling illnesses occurred with greater frequency among children under 10 years of age than among persons in any subsequent period except old age.

The duration of the average disabling illness was found to be lowest among children, and, except in the period of infancy, the rate of recovery from illness was notably higher among children than among adults.

Four in every 5 disabling illnesses occurring among children under 15 years of age were due to acute communicable diseases or diseases of the respiratory tract; these diseases accounted for over half of the disability experienced by the average child in the twelve month period. Broadly considered, the control of the acute communicable diseases and diseases of the respiratory tract represents the major problem in the field of child health, since many of these diseases are preventable.

In general, a smaller proportion of the disabling illnesses of children than of adults received care from a physician in the home, clinic or physician's office but the age variation in the proportion of illnesses so attended was not marked except in cities with a population under 25,000.

The average number of consultations with a physician per patient was lowest among children. Cases of the acute communicable diseases and minor diseases of the respiratory tract accounted for almost three fourths of the child patients attended by a physician outside the hospital and for about one half of the physicians' services to children; these diseases, on the average, do not require intensive medical supervision.

Among both children and adults, the proportion of disabling illnesses receiving bedside nursing care was relatively low compared with the proportion receiving care from a physician. The proportion of illnesses attended by a visiting nurse was highest in childhood and decreased with age; private duty nursing care showed a reversal of this relation. Disabling illnesses due to the communicable diseases and minor diseases of the respiratory tract accounted for the majority of child patients receiving bedside nursing care.

In the cities with population of 100,000 and over, 1 in 24 children under 15 years of age had been a hospital patient in the year of survey; in the cities of population under 25,000, the ratio was 1 to 38. Over half of the hospital cases among children were surgical; diseases of the tonsils and adenoids alone accounted for about two fifths of the cases. In the large cities, the frequency of hospitalization in cases of the

acute communicable diseases was relatively high; hospital care in these cases was infrequent in the cities of population under 25,000.

Among children in families on relief and in self-sustaining families with incomes up to the $2,000 level, the proportion of disabling illnesses receiving care from a physician outside the hospital was notably lower than in families with incomes of $3,000 and over. This relation was observed consistently in the large, medium-sized and small cities.

The nature of the association between family income and the receipt of hospital care by children showed variation with the size of the cities. In the small cities, the proportion of the illnesses of children receiving hospital medical care increased progressively as family income increased; in the large cities, this association was reversed. It was found that varying practice in large and in small cities in the hospitalization of medical, as distinguished from surgical, illnesses among children largely accounted for this difference.

In each income class, the illnesses of children showing the highest proportion of cases in which no medical care was given were those due to the communicable diseases and to minor diseases of the respiratory tract, exclusive of tonsillitis; but the proportion of cases in which these diseases were attended increased with increasing family income.

For all of the diseases of childhood the illnesses of children attended by a physician outside the hospital received more intensive care in families with high than in those with low incomes. The average number of hospital days per child patient, however, was consistently higher among families with low incomes.

FACTORS AFFECTING RETENTION OF NITROGEN AND CALCIUM IN PERIOD OF GROWTH

II. EFFECT OF THYROID ON CALCIUM RETENTION

J. A. JOHNSTON, M.D.
AND
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DETROIT

The role of the thyroid in calcium metabolism has been investigated by Aub and others [1] with low calcium intake, and they have concluded that in cases of exophthalmic goiter and hyperfunctioning adenoma the excretion is increased and that in cases of myxedema it is markedly diminished and that the giving of thyroid to normal persons and to patients with myxedema increases the excretion. They expressed the belief that this was due to a direct stimulating effect on the calcium deposits in the bones. Their data on the patients with exophthalmic goiter were striking and definite; the figures on those with myxedema left some doubt. Thus, even allowing for the variation inherent in short term balances, when isolated figures are picked at varying metabolic levels the following are difficult to reconcile with their thesis:

Subject	Total Calcium Output, Mg.	Basal Metabolic Rate
Susan F.	0.68	—39
	0.42	— 5
Jennie W.	1.02	—39
	0.61	+ 0
Mabel M.	0.52	—29
	0.45	—17
	(Subsequently the output rose to 0.76 mg., while the basal metabolic rate went to —4%)	
John F.	0.51	—37
	0.51	— 9
	(Intermediate figures were 0.49 and 0.71 mg.)	

From the Department of Pediatrics, Henry Ford Hospital.

Read at the annual meeting of the Society for Pediatric Research, Skytop, Pa., April 27, 1939.

1. Aub, J. C.; Bauer, W.; Heath, C., and Ropes, M.: Studies of Calcium and Phosphorus Metabolism: III. The Effects of the Thyroid Hormone and Thyroid Disease, J. Clin. Investigation 7:97 (April) 1929.

Although many of the balances do show an increased output, they do not impress us as being proportionate to the increase in the metabolic rate. While the technic employed has advantages for certain types of investigation, from the standpoint of measuring utilization and retention —involving as they do the question of absorption—the low intake method would seem to us to have definite limitations. If the effect of any variable introduced into the problem could be shown to be related to absorption and be reflected largely in the fecal calcium level, this effect would be obscured by a low intake.

The work of Aub and his associates was reviewed by Hansman and Wilson,[2] who used a higher intake. Their findings for patients with elevated metabolism agreed with those of the aforementioned authors, although the two groups of experimenters differed sharply in their interpretation of them, Hansman and Wilson expressing the belief that an associated hyperparathyroidism is the direct cause of the excessive mobilization and that hyperthyroidism per se has no specific effect on calcium and phosphorus metabolism. They gave no data on the effect of the administration of thyroid to patients with hypothyroidism. Using the technic of Aub and his associates, Cope and Donaldson [3] studied a case of hypoparathyroidism with hyperthyroidism. Since a marked increase in the negative balance of calcium and phosphorus beyond normal was found with hyperthyroidism, even though the signs of diminished parathyroid activity continued, they concluded that the changes could not be related to overactivity of the parathyroids. Pugsley and Anderson [4] expressed the belief that their work showing an increased excretion in rats following administration of thyroid confirmed the thesis of Aub and his associates that the increase was a result of fecal loss and did not correlate well with the basal metabolic rate. They used 2 grains (0.13 Gm.) a day, a large dose.

The prompt growth of the cretin following thyroid medication and the acceleration of growth in the bones, evident in the epiphyses, would imply an improvement in the utilization of calcium referable to thyroid that would make its role in calcium metabolism, at least in the period of growth, appear anabolic. But again it may not be a question of the

2. Hansman, F. S., and Wilson, F. H.: Calcium and Phosphorus Metabolism in Diseases of the Thyreoparathyreoid Apparatus: I. Calcium, Phosphorus and Total Metabolism in Hyperthyreoidism, and the Part Played by the Parathyreoid Glands, M. J. Australia **1**:37 (Jan. 13) 1934.

3. Cope, O., and Donaldson, G. A.: Relation of Thyroid and Parathyroid Glands to Calcium and Phosphorus Metabolism: Study of a Case with Coexistent Hypoparathyroidism and Hyperthyroidism, J. Clin. Investigation **16**:329 (May) 1937.

4. Pugsley, L. I., and Anderson, E. M.: The Effect of Desiccated Thyroid, Irradiated Ergosterol and Ammonium Chloride on the Excretion of Calcium in Rats, Biochem. J. **28**:754, 1934.

period of growth but of the rate of oxidation, and departures from the optimum rate, either upward or downward, may not be consistent with normal retention. Since the negative balances of hyperthyroid patients seem largely referable to increased urinary output and the improved balances with thyroid administration which we feel are shown in the studies here to be reported are accounted for by diminished fecal loss, it is evident that the latter might not be noticeable when the intake is low.

The subjects were all in the period of growth. The technic involved continuous balance studies; the diets administered were comparatively high in calcium, phosphorus and nitrogen and calorically adequate for growth.

METHOD

The methods for determining nitrogen and the basal metabolic rate were described in the first article of this series.[5] Calcium was precipitated from solutions of the ash of food, feces or urine and, as the oxalate, was titrated with potassium permanganate. The conditions of precipitation were those of Shohl and Pedley.[6] In determinations on urine and stools the oxalate was separated and washed by centrifuging and decanting. In determinations on food, filtration and washing were done on Jena sintered glass crucibles.

The initial steps for these determinations varied with the material. The sample of food consisted of an average day's diet, prepared by weighing out each day for six days an aliquot equal to one sixth of all food received by the patient. This material was hydrolyzed with hydrochloric acid and heated until it was satisfactory for sampling. After dilution to 4,000 cc., 25 cc. samples were taken for calcium analyses and 2 cc. samples for nitrogen analyses. For calcium the samples were ashed by repeated evaporation on the sand bath with nitric and with perchloric acid in 500 cc. pyrex beakers of either the Berzelius tall or the Phillips conic type. Heating was carefully controlled. The final ash was dissolved with hydrochloric acid, and the precipitation and the final titration were carried out in the original beaker. The feces were collected directly in 6 inch (15 cm.) French glass jars and were put into hydrochloric acid. After three days a suspension was prepared in the same manner as with food except that the volume was made up to 2,000 cc., and 5 cc. samples were ashed in 125 cc. beakers. Samples of urine were ashed in the same way. All analyses for calcium and nitrogen were made in duplicate.

FINDINGS

Hyperthyroidism with Postoperative Hypothyroidism.—SUBJECT 1.—V. C. (fig. 1 and table 1), a girl aged 15 years, was brought to the clinic for a tonsillectomy, with a history of repeated sore throats, but because of a rapid pulse and clinical signs of hyperthyroidism she was admitted for study. Her initial basal metabolic rate was + 41 per cent. After a course of iodine medication, high caloric diet and rest in bed, surgical intervention was decided on. The operative steps are recorded in table 1,

5. Johnston, J. A., and Maroney, J. W.: Factors Affecting the Retention of Nitrogen and Calcium in the Period of Growth: I. Effect of Thyroid on Nitrogen Retention, Am. J. Dis. Child. **58**:965 (Nov.) 1939.

6. Shohl, A. T., and Pedley, F. G.: A Rapid and Accurate Method for Calcium in Urine, J. Biol. Chem. **50**:537 (Feb.) 1922.

TABLE 1.—*Effect of Thyroid on Calcium Retention of V. C.* (Hyperthyroid)*

Standard Diet 1-6-2,200 Calories

Comment	Six Day Periods No.	Dates 1937	Nitrogen, Mg. Output	Intake	Balance	Calcium, Mg. Urine	Stool	Total Output	Intake	Balance	Basal Metabolic Rate	Total Calories	Weight, Pounds
	1	8/14–19	11.96	12.84	+0.88						+45	2,016	94½
											+56	2,145	93½
	2	8/20–25	12.97	11.55	—1.42	0.361	1.038	1.399	1.253	—0.146	+48	2,021	90¼
	3†	8/26–28	12.98	12.59	—0.39	0.450	1.140	1.590	1.170	—0.420	+45	1,962	88¾
Ligation 8/30; right lobectomy 9/10													
	4	9/17–22	10.32	9.97	—0.35	0.315	1.063	1.378	1.134	—0.244	+ 1	1,370	88
Peritonsillar abscess	5	10/18–22	12.85	11.99	—0.86	0.133	1.513	1.646	1.383	+0.263	+21	1,710	98¼
	6	10/23–28	11.77	6.97	—4.80	0.090	0.948	1.038	1.389	+0.351			
	7	10/29–11/3	10.47	9.21	—1.20	0.191	0.956	1.147	1.083	—0.065			
											+14	1,558	91
	8	11/ 4– 9	10.27	10.40	—0.13	0.268	1.339	1.607	0.845	—0.762	+ 7	1,487	93
	9	11/10–15	7.53	8.29	+0.76	0.111	1.070	1.181	1.108	—0.073	+ 8	1,522	94
Tonsil and adenoid operation 1/13											+ 8	1,494	92
	10	11/16–21	7.55	9.81	+2.26	0.160	1.071	1.232	1.079	—0.153	+ 5	1,455	91
											+ 3	1,428	92
	11	11/22–27	8.27	11.52	+3.25						+ 3	1,419	92½
Left lobectomy 1/5		1938											
	12	1/ 9–14	9.33	11.31	+1.98	0.079	1.060	1.139	1.196	+0.057			
	13†	1/15–17	8.06	11.78	+3.72	0.258	1.537	1.795	1.143	—0.652	—14	1,237	100
											—20	1,198	114
	14	2/17–22	10.76	13.68	+2.92	0.012	1.034	1.046	1.261	+0.215	—15	1,238	117½
											—19	1,282	115
B. W. thyroid, 1½ grains a day, 3/19													
	15	2/23–28	9.04	13.29	+4.25	0.014	0.495	0.509	1.256	+0.747	—14	1,267	115
	16	3/ 1– 6	10.51	13.16	+2.65	0.009	0.645	0.654	1.265	+0.611	—17	1,256	115¼
B. W. thyroid, 2 grains a day, 2/24													
	17	3/ 7–12	10.56	13.17	+2.61	0.027	0.795	0.822	1.245	+0.423	—18	1,208	114½
											—10	1,316	114½
	18	3/13–18	11.08	12.03	+0.95	0.027	0.703	0.730	1.155	+0.425	—10	1,317	115
B. W. thyroid, 3 grains a day, 3/7													
	19	3/19–24	7.41	10.94	+3.53	0.011	0.610	0.621	1.089	+0.468	— 5	1,407	114¾

* Born April 30, 1922; admitted Aug. 6, 1937; weight 94½ pounds (42.8 Kg.); estimated normal weight 125 pounds (56.7 Kg.); height 66 inches (168 cm.).
† Three day period.

as are the time of a severe bilateral peritonsillar abscess and of tonsillectomy. The various operative procedures required interruptions in the continuity of the balance studies; these are indicated in figure 1 by breaks in the cross hatching. In addition to the ligation of the vessels, the surgical procedure on the thyroid consisted in the removal of approximately 14 Gm. from the right lobe and 20 Gm. from the left. On section the material was classified as showing third degree hyperthyroidism and second degree hyperplasia. On Jan. 14, 1938, the blood calcium content was 9.8 mg. per hundred cubic centimeters and the phosphorus content 3.79 mg.

The data bring out well the negative balances associated with the hyperthyroid state. The contrasting side of the picture following the

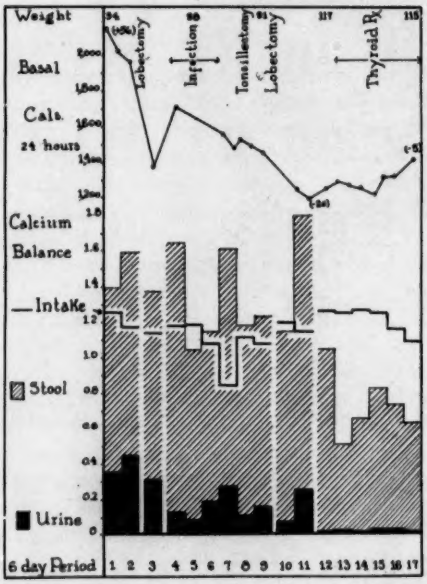

Fig. 1 (V. C., hyperthyroid).—Results of balance studies. The solid black portion represents the urinary output of calcium in grams per day per six day period; the cross hatching, the calcium in the stool and the single black line, the intake. Breaks in the continuity of the chart attended operative procedures, and the exact time covered is indicated in table 1.

bilateral lobectomy, however, showed that, while the urinary loss was offset, the fecal loss was still high and was strikingly reduced when, with the basal metabolic rate at —20 per cent, thyroid medication was instituted. A single six day balance was positive just before thyroid medication was begun, but the value was doubled with the treatment. It is this economy referable to the fecal fraction that we feel is missed with the low intake technic.

The study suggested that in this adolescent optimal utilization of calcium occurred when the metabolism approached normal, that the hyperthyroid state is inconsistent with normal retentions, as a result of

high urinary excretion, and that the hyperthyroid state likewise does not favor it, apparently as a result of high fecal loss.

Cretinism.—SUBJECT 2.—The history of D. E. (fig. 2 and table 2) was recorded in the first article of this series,[5] with the data on her nitrogen metabolism. The calcium balance study was continued without interruption only during one course of thyroid administration, for a total of fifty-four days. For the first twelve days there existed a strongly negative balance, which was then reduced as thyroid was given and even before any appreciable rise had occurred in the metabolic rate.

That such a result should occur might have been expected from the usual clinical observation of the cretin, but the inference from work on

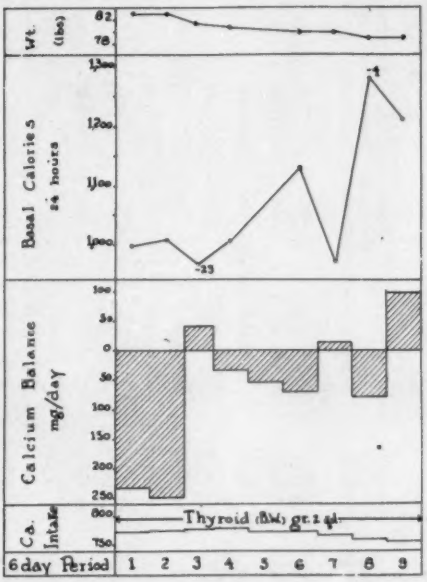

Fig. 2 (D. E., cretin receiving thyroid).—The balance alone is charted. As will be seen in table 2, no significant change occurred in the urinary calcium.

the adult myxedematous patient is that the low metabolism of this condition is associated with high retention.

Normal Thyroid Function.—SUBJECT 3.—The history of R. B. (fig. 3 and table 3) is likewise recorded with the study of his nitrogen metabolism,[5] and the results for calcium paralleled those for nitrogen.

Two slight infections of the upper respiratory tract accompanied with some fever but not resulting in any refusal of food complicated the period of high thyroid intake.

Whether the patient is considered as having "masked hypothyroidism" or as normal, it is apparent that a small amount of thyroid produced an anabolic effect on the calcium balance, and a larger amount reduced the balance.

TABLE 2.—*Effect of Thyroid on Calcium Retention of D. E.* (Cretin)*

No.	Date 1937	Calcium, Mg.					Basal Metabolic Rate	Respiratory Quotient	Calories per Square Meter per Hour	Basal Calories	Height, Inches	Weight, Pounds
		Urine	Stool	Total Output	Intake	Balance						
1	3/ 3- 8	0.043	0.963	1.006	0.772	—0.233	—21	0.88	34.7	998	54½	83
2	3/ 9-14	0.054	0.970	1.024	0.775	—0.249	—18	0.89	35.3	1,008	54½	83
3	3/15-19	0.044	0.693	0.737	0.778	+0.041	—23	0.88	33.9	967	54½	81½
4	3/20-25	0.037	0.775	0.812	0.779	—0.033	—19	0.84	35.5	1,006	54½	80¾
5	3/26-31	0.043	0.785	0.828	0.775	—0.053						
6	4/ 1- 6	0.034	0.811	0.845	0.775	—0.070	—10	0.86	40.0	1,130	54½	80
7	4/ 7-12	0.026	0.756	0.782	0.768	+0.014	—22	0.81	34.3	972	54½	80
8	4/13-18	0.013	0.827	0.840	0.764	—0.076	— 4	0.84	46.1	1,284	54½	79
9	4/19-24	0.019	0.641	0.660	0.758	+0.098	— 2	0.77	43.5	1,211	54½	79

* Born in 1925; aged 12 years; height 54½ inches (138 cm.); estimated normal weight 71 pounds (32.2 Kg.). Thyroid (Burroughs, Wellcome), 2 grains a day, and special diet no. 2, consisting of 1,400 calories (15 per cent, 52.4 Gm., of protein, 35 per cent, 54.4 Gm., of fat and 50 per cent, 175 Gm., of carbohydrate) were given throughout this study.

TABLE 3.—*Effect of Thyroid on Calcium Retention of R. B.**

Six Day Periods Comment	No.	1938–1939 Dates	Standard Experimental Diet 1-6, 1,900 Calories with Klim — Calcium, Mg. Av. Urine	Av. Stool	Total Output	Intake	Balance	Basal Metabolic Rate	Respiratory Quotient	Calories per Square Meter per Hour	Total Calories	Weight, Pounds	Height, Inches
	1	10/21–10/25	0.091	0.758	0.849	1.220	+0.371						
	2	10/26–10/31	0.058	0.928	0.986	1.195	+0.207						
	3	11/ 1–11/ 6	0.015	1.124	1.139	1.170	+0.031	— 6	0.87	48.5	1,036	49¾	49
Control Period					Mean	balance	+0.186						
Thyroid (B. W.), 1 grain a day, 11/8-11/30													
	4	11/ 7–11/12	0.115	0.950	1.065	1.170	+0.105	— 8	0.84	47.3	1,021	50½	49
	5	11/13–11/18	0.118	0.931	1.049	1.110	+0.061	— 8	0.84	47.3	935	51½	49
	6	11/19–11/24	0.077	0.830	0.907	1.120	+0.203	— 4	0.79	49.1	1,060	51½	49
	7	11/25–11/30	0.110	0.823	0.933	1.190	+0.257	— 6	0.83	48.3	1,042	51	49
					Mean	balance	+0.159						
Thyroid (B. W.), 2 grains a day, 12/1-12/18													
	8	12/ 1–12/ 6	0.120	0.925	1.045	1.190	+0.145	— 5	0.82	49.0	1,070	52½	49
	9	12/ 7–12/12	0.115	0.820	0.935	1.210	+0.275	— 9	0.77	46.5	1,014	52½	49
	10	12/13–12/18	0.115	0.785	0.900	1.230	+0.330						
					Mean	balance	+0.250						
Thyroid (P. D.), 2 grains a day, 12/19-1/11													
	11	12/19–12/24	0.095	0.884	0.979	1.120	+0.141	— 9	0.76	47.3	1,032	52	49
	12	12/25–12/30	0.079	1.067	1.146	1.065	—0.081	— 2	0.80	50.6	1,093	51½	49
	13	12/31– 1/ 5	0.115	0.950	1.065	1.120	+0.055	+ 1	0.83	52.2	1,126	50	49
	14	1/ 6– 1/11	0.101	1.025	1.126	1.145	+0.019	+ 2	0.83	52.8	1,140	50¾	49
					Mean	balance	+0.033						
Thyroid (P. D.), 3 grains a day, 1/12-1/29													
	15	1/12– 1/17	0.111	1.063	1.174	1.260	+0.086	+ 5	0.79	53.9	1,150	50	49
	16	1/18– 1/23	1.111	0.908	1.019	1.165	+0.146						
	17	1/24– 1/29	0.132	0.969	1.101	0.980	—0.121	+ 5	0.79	54.5	1,175	51¼	49
					Mean	balance	+0.118						
	18	1/30– 2/ 4	0.103	1.020	1.123	1.190	+0.067	+12	0.76	57.8	1,231	49½	49
	19	2/ 5– 2/10	0.058	0.925	0.983	1.150	+0.167	— 2	0.82	50.4	1,077	50	49
	20	2/11– 2/16	0.063	0.770	0.833	1.110	+0.277	— 9	0.85	46.6	1,006	51	49
					Mean	balance	+0.170						

* Born Dec. 6, 1929; weight 46 pounds (20.9 Kg.); height 49 inches (124.5 cm.); estimated normal weight 55 pounds (24.9 Kg.).

Osteochondritis Juvenilis (Legg-Perthes Disease).—Subject 4.—W. A. (fig. 4 and table 4), a boy aged 9 years, was seen in March 1937 because of a limp. Roentgenograms revealed a normal hip, with some question of "slight osteoporosis." Three months later there was definite evidence of some flattening of the head of the femur and decreased density in the neck, with narrowing of the joint space. The tuberculin test was negative. In August, when the study started, the basal metabolic rate was determined on seven occasions in the control period, being —3, —6, —7, —8, —11, —13 and —17 per cent. The figures in table 4 are averages of all determinations made within a given six day period. Roentgenograms of the wrist showed a normal osseous age.

The first diet selected seemed inadequate and was accompanied with negative nitrogen and calcium balances, a fall in metabolic rate and a slight loss of weight.

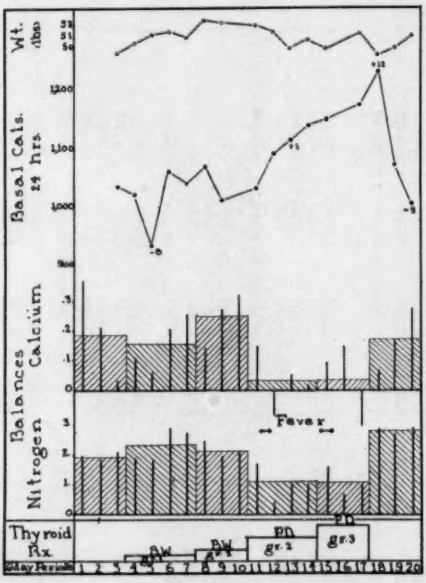

Fig. 3 (R. B., normal boy receiving small and large amounts of thyroid).—An increase in retention is noted with the small amounts and a decrease with the large. Single bars represent six day periods; the cross hatched blocks represent an average of all the six day periods contained within them. *BW* refers to Burroughs, Wellcome thyroid, and *PD* to Parke, Davis preparations.

Accordingly, increases were made in the intake of calories, nitrogen and calcium. The nitrogen balance became positive, but the calcium balance was not altered.

The nitrogen balance showed a slight falling off after both the first and the second series of injections of an anterior pituitary extract said to contain the growth-promoting principle (antuitrin G). The calcium balance, which was markedly negative, became less negative after the first series, but showed little change with the second series. A positive balance was promoted by thyroid therapy, which was interrupted by the occurrence of acute follicular tonsillitis with fever. After its subsidence the balance again became positive.

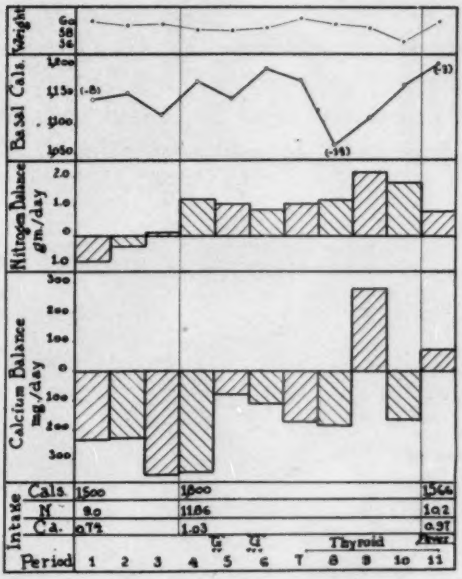

Fig. 4 (W. A., with osteochondritis juvenilis).—Each cross-hatched block represents a six day period. The observation covers an uninterrupted period of sixty-six days. An increase in the intake of nitrogen and of calcium in the fourth period resulted in a positive nitrogen balance but had no effect on the calcium balance. *G* refers to an anterior pituitary extract said to contain the growth-promoting principle (antuitrin G). *Thyroid* indicates 2 grains (0.13 Gm.) daily of the Burroughs, Wellcome preparation.

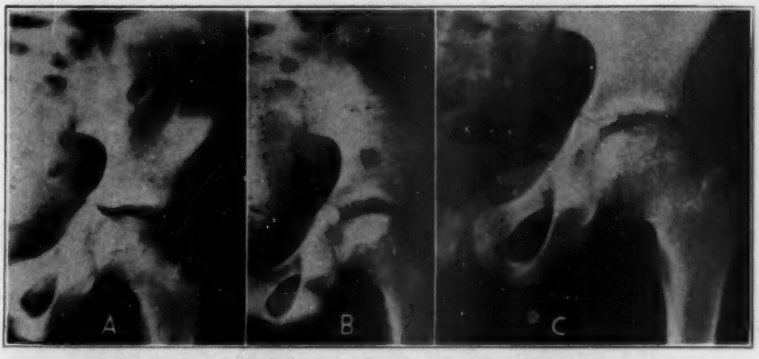

Fig. 5.—Roentgenograms of the hip of W. A., with osteochonditis juvenilis: *A*, March 11, 1937; *B*, Aug. 19, 1937; *C*, Nov. 15, 1938.

TABLE 4.—*Effect of Thyroid on Calcium Retention of W. A.,* with Perthes Disease*

Standard Experimental Diet 1-6, 1,500 Calories

1937 Dates	Nitrogen, Mg. Output	Intake	Balance	Calcium, Mg. Output	Intake	Balance	Basal Metabolic Rate	Respiratory Quotient	Basal Calories	Weight, Pounds	Height, Intake	Comment
8/23–25	9.43	9.00	−0.43	0.930	0.738	−0.192	−17	0.79	1,045	61	55¼	
8/26–28	10.31	9.00	−1.31	1.010	0.738	−0.272	− 3	0.77	1,238	60	55¼	
8/29–31	9.95	9.00	−0.95	1.106	0.738	−0.368	− 7	0.77	1,178	59¼	55¼	
9/ 1– 3	8.77	9.00	+0.23	0.823	0.738	−0.085	−11	0.83	1,125	59½	55¼	
9/ 4– 6	8.93	9.00	+0.07	0.666	0.738	−0.072						
9/ 7– 9	8.87	9.00	+0.13	1.510	0.738	−0.772	−13	0.80	1,116	60	55¼	
		Mean balance	−0.37			−0.204						
Diet 1,500 calories 1,800 calories												
9/10–14	10.43	11.86	+1.43	1.370	1.030	−0.340	− 6	0.81	1,183	59	55¼	
							− 8	0.83	1,159	59	55¼	
9/15–16	10.76	11.86	+1.10	1.082	1.030	−0.052	−10	0.83	1,141	59	55¼	Antuitrin G, 15 cc. a day
9/17–20	10.72	11.86	+1.14	1.285	1.030	−0.255	−11	0.82	1,126	59	55¼	
							− 8	0.85	1,166	59	55¼	
9/21–23	11.13	11.86	+0.75	1.319	1.030	−0.289						Antuitrin G, 15 cc. 9/21 and 22 Antuitrin G, 5 cc. 9/23
9/24–27	10.92	11.86	+0.94	1.082	1.030	−0.052	−13	0.79	1,096	59½	55¼	
9/28–30	10.35	11.86	+1.51	1.439	1.030	−0.409						
10/ 1– 3	11.11	11.86	+0.75	0.970	1.030	+0.060	− 6	0.85	1,173	61	55¼	
10/ 4– 6	10.37	11.86	+1.49	0.951	1.030	+0.079	−17	0.82	1,028	60	55¼	
10/ 9– 9	11.70	11.86	+0.16	1.423	1.030	−0.393	−11	0.81	1,107	60	55¼	
		Mean balance	+0.97			−0.137						
10/10–12	9.79	11.86	+1.33	0.921	1.030	+0.109	−12	0.87	1,100	59½	55¼	Thyroid (B. W.), 2 grains a day
10/13–15	9.48	11.86	+2.38	0.580	1.030	+0.450	− 9	0.80	1,123	59	55¼	
10/16–18	8.94	11.86	+2.92	1.163	1.030	−0.133						
10/19–21	11.11	11.86	+0.76	1.230	1.030	−0.200	− 4	0.88	1,168	57	55¼	
10/22–24	8.49	11.86	+3.37	0.958	0.937	−0.021	− 3	0.82	1,213	60½	55¼	
10/25–27	10.27	11.86	+1.59	0.828	1.002	+0.174						
		Mean balance	+2.06			+0.060						

* Age 8 years; height 53½ inches (136 cm.); weight 60¼ pounds (27.3 Kg.); estimated normal weight 68½ pounds (31 Kg.).

It has been suggested elsewhere [7] that osteochondritis juvenilis improves with preparations containing the "growth hormone"—hence the trial of such an extract here. Our failure with it may have been the result of too small a dose of the extract, but the completely negative result of the second trial is an experience we have now had a number of times, and whether because of the development of antihormone or not, makes us question the usefulness of the material when repeatedly injected.

Fig. 6.—Roentgenogram of the wrists of W. A. (chronologic age 8 years; osseous age normal).

Reilly and Smyth [8] have described what they term "cretinoid epiphyseal dysgenesis" as a condition to be distinguished from Calvé-Legg-Perthes disease, stating that it responds to thyroid medication while osteochrondritis juvenilis does not; this particular case, however, seemed to us a typical instance of the latter condition. The lesion was unilateral; a destructive process, not noted in the first roentgenogram, was evident in the second, and the osseous age according to the Todd standard

7. Engelbach, W., and Schaefer, R. L.: Endocrine Dwarfism, Endocrinology **18**:387 (May-June) 1934.

8. Reilly, W. A., and Smyth, F. S.: Cretinoid Epiphyseal Dysgenesis, J. Pediat. **11**:786 (Dec.) 1937.

appeared to correspond with the chronologic. Those authors spoke incidentally of using calcium to replace that "lost by the action of thyroid substance" used therapeutically, implying that this is the usual effect of thyroid. Cavanaugh and others [9] have reported 5 cases of osteochondritis with evidence of hypothyroidism in which epiphysial structure responded well to thyroid therapy, but they gave no data on calcium balance. The basis for the diagnosis of hypothyroidism was chiefly osseous age. Benjamin and Miller [10] gave an excellent review of the whole problem. Whether the condition was truly osteochondritis juvenilis or cretinoid dysgenesis, the fact remains that the administration of thyroid was followed by an increased rather than a decreased retention of calcium, which supports the thesis of this paper and is the reason for inclusion of the case here.

SUMMARY AND CONCLUSIONS

We present studies of the calcium balance of 4 children: A child with hyperthyroidism who underwent ligation and bilateral lobectomy, bringing the metabolism to subnormal levels and requiring thyroid medication; a cretin receiving thyroid; a normal boy receiving thyroid, and a boy with osteochondritis juvenilis who received thyroid. In all cases optimal retentions were noted when the metabolism approached normal.

It is concluded that in the period of growth thyroid in doses small enough so that the basal metabolism is not stimulated beyond normal increases retention. Beyond this point retention is diminished.

Dr. Arthur B. McGraw performed the lobectomy and Dr. Roy D. McClure the ligation in the case of V. C.; Dr. C. L. Mitchell supervised the orthopedic care of W. A.; Dr. Frank Hartman supervised the determinations of basal metabolism and Dr. O. H. Gaebler the determinations of calcium and nitrogen. Dr. Gaebler also wrote the paragraph on methods.

9. Cavanaugh, L. A.; Shelton, E. K., and Sutherland, R.: Metabolic Studies in Osteochondritis of the Capital Femoral Epiphysis, J. Bone & Joint Surg. **18**: 957 (Oct.) 1936.

10. Benjamin, B., and Miller, P. R.: Hypothyroidism as a Cause of Disease of the Hip, Am. J. Dis. Child. **55**:1189 (Jan.) 1938.

THE PLAY-INTERVIEW: A METHOD OF STUDYING CHILDREN'S ATTITUDES

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Parents and others who have observed children playing with dolls have heard them repeat the scoldings which they have received, as well as voice their own interests and attitudes. A glimpse could thus be obtained of the personal life of the child.

It has been possible to utilize these natural tendencies to self-expression in experimental play situations. The method of the play-interview, which I developed at the Children's Psychiatric Clinic (conducted by Dr. Leo Kanner) of the Harriet Lane Home, Johns Hopkins Hospital, serves to supplement other clinical methods and contributes facts concerning the imaginative and emotional aspects of the child's behavior.

The procedure provides opportunities for the child to express his feelings and thoughts through the medium of dolls as if they were responsible for all that is said and done. Thus the child, as an impartial spectator, can view objectively what is going on at the same time that he is actively participating in an intimate discussion of his own attitudes. It is not the child himself but the doll who is afraid of the dark. It is not he who is jealous or hates, but the doll character. Therefore, he can describe the motives and imaginations that may help to explain the doll's behavior and consequently his own. Toy furniture and dolls representing various characters, such as parents, teachers and siblings, are used during the play session, and various "sets" are arranged by the physician as on a miniature stage.

The child is given an opportunity to play freely with the dolls, but the emphasis in this procedure is placed on planned play situations, which usually are repeated at weekly intervals at first and thereafter as frequently as desirable. Such problems as car sickness,[1] fear states,[2]

Presented at the sixty-fourth meeting of the Interurban Clinical Club, April 15, 1939.

From the Children's Psychiatric Clinic of the Harriet Lane Home for Invalid Children, Johns Hopkins Hospital.

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1. Conn, J. H.: A Psychiatric Study of Car Sickness in Children, Am. J. Orthopsychiat. **18**:130-141 (Jan.) 1938.

2. Conn, J. H.: Child Speaks to the Psychiatrist: An Introduction to the Method of the Play-Interview, Occup. Therapy **17**:231-244 (Aug.) 1938.

sibling jealousies, reactions to parental undersolicitude or oversolicitude and more recently the sex attitudes and sex awareness of the child[3] have been studied in an unbiased, self-critical manner by a series of repeated play experiments.[4]

The case of Edward, aged 7, with an intelligence quotient of 100, presents the problem of car sickness and its personal setting. This boy is fairly representative of the group of 83 car sick children who have been studied since 1933. Edward was first seen on April 24, 1937. On that occasion his mother stated:

> He is so sick on the cars. Any time he gets on a car or even in a machine, he gets sick at the stomach and he gets vomiting spells. After that he is relieved and is all right. I take him off the car just before he gets sick, and I take a taxi. I am afraid to get on the car with him. He was about 3 when it began, and he would get white on an automobile or streetcar. He gets sick on the way in to Baltimore, and on the way back he never vomits; he just says he feels "bad." He never turns white.

The mother also was carsick. She was afraid to remain alone in the house and was frightened by thunderstorms. She first began to take long car rides when she was 14. She had been working at her first job in the city and had to come in from one of the near-by towns. She stated that she could remember her attitude to these rides:

> I was afraid that I might get lost in the big city. I was scared going and coming alone. It was really the feeling I wouldn't get there safe and get back safe. . . . My nerves have always been bad.

She went on to describe Edward as a jumpy, fidgety child who was fussy about his food and unable to take care of himself in a fight. Edward's attitude to riding on a car was studied by placing a boy doll in a toy streetcar and pushing the car to and fro. He was then told:

> *Q.*—This boy is riding on the streetcar. How does he feel? *A.*—His stomach hurts.
>
> *Q.*—What does he think? *A.*—He will get sick. He thinks he has to go to the hospital, that he has to stay there for a long time.
>
> *Q.*—What else? *A.*—There might be a wreck and scare him. That's the only ideas I have.
>
> *Q.*—And? *A.*—There might be a bump in. There'd be a wreck. He'd be sitting in the front seat, and he'd be sick. . . .
>
> *Q.*—It's whose idea? *A.*—It's the boy's.
>
> *Q.*—Not the? *A.*—Streetcar. It's the boy's fault. He was thinking. No wonder I get sick. I seen cars turn curves sudden. Heck, one time I kept on looking at the pictures on the streetcar and I didn't get sick.

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3. Conn, J. H.: Sex Attitudes and Sex Awareness in Young Children, Child Study **16**:86-106 (Jan.) 1939.

4. Conn, J. H.: The Child Reveals Himself Through Play, the Method of the Play-Interview, Ment. Hyg. **23**:49-69 (Jan.) 1939.

Q.—Why? *A.*—I didn't think in case of there'd be a wreck, or if the car ran over a stone and turned over. . . .

Q.—Coming back why doesn't he get sick? *A.*—He is thinking of something else, like movies (if it's a Saturday) or playing ball. I think of having a good time (when coming home).

Q.—When you leave home? *A.*—I'm going to town, and that means a whole bunch of machines and traffic.

Q.—Coming home? *A.*—There is a little bit of traffic. You don't have to worry about wrecks.

Q.—Now you can ride on cars. It's not the streetcar. *A.*—There is not always going to be a wreck every day.

The second play-interview took place a week later (May 1). The mother stated:

We made it all right. He did get sort of white coming up this morning. He acted like he was trying to fight it. We never could get up this far with him. We always had to get off at Fifteenth Street (which is half the distance from their suburban home). We didn't have any trouble on the way home last week. We never have any trouble with him going home. . . .

Edward was asked:

Q.—How have you been on streetcars? *A.*—I wasn't sick any. . . .

Q.—Why didn't you get sick? *A.*—I don't know.

A boy doll was placed on a toy streetcar and Edward was asked:

Q.—He is riding on the car. How does he feel? *A.*—A little bit sick and a little bit not.

Q.—What is he thinking of? *A.*—He thinks he is going to get sick maybe.

Q.—He rides along, and then he thinks? *A.*—That there is something to be afraid of.

Q.—Such as? *A.*—A wreck.

Q.—And then he thinks? *A.*—That if there is going to be a wreck, he will get sick and scared. . . .

Q.—Now he knows? *A.*—Better. It wasn't the streetcar's fault, and it won't be his fault if he doesn't think of it, and if he does think of it, it will be his fault.

A week later Edward was interviewed for the third time (May 8). His mother reported:

He has been on a car once this week; that was today. He did fine. He didn't even turn white. He was a little fidgety, but it was an hour's ride. I never could get any further than halfway since he has been 3 or 4 years of age. He never could ride long distances.

Edward came into the playroom and said:

I never got sick ever since the first time you told me about it.

Q.—Why? *A.*—'Cause I look at different pictures on the streetcars and get a box of cakes and eat them, or I look out of the window and think of different things.

The toy streetcar with the boy doll in it was again placed before the patient:

Q.—How does he feel on the streetcar? *A.*—When he is going up, he thinks he is going to get sick. When he is going to his grandmother's, he is thinking that he is going to get sick in the middle of town. He wouldn't be close to his grandmother's and he wouldn't be close to home.

Q.—Now he knows? *A.*—That it's all his fault. . . .

Q.—Now you can? *A.*—Ride on the streetcars.

Q.—It's not the streetcar—? *A.*—And I can go to my grandmother's and all. It's what the little boy thinks.

The fourth play-interview occurred two weeks later (May 22). The mother stated that he had taken two long car trips without any return of his nausea or vomiting. Edward was still mildly apprehensive about the cars during this interview. He was asked:

Q.—How does he (the boy doll) feel on the streetcar now? *A.*—He feels all right when coming back. When he is going up, he don't feel all right. . . . The streetcar might turn over, might run over somebody, might run into a pole.

Again Edward was able to say that it was the boy's fears which helped to upset him.

He was seen for the sixth time two months later (July 24). The mother reported that Edward had been on several streetcar rides and on a long automobile trip and had not vomited or complained in any way. Previously he had been just as upset while riding in an automobile as he had been in a streetcar.

Edward was interviewed seven weeks later (September 11). The mother reported:

Everything is O. K. He doesn't get sick on the car any more. We took him on our vacation and drove through Virginia. He wasn't sick in the machine. Before he was sick in the machine like he was in the car. He'd get real white and then vomit. This would happen after a short ride as well as a long ride. *(And now?)* He seems all right. We take him most anywhere and nothing happens.

Edward was seen again on Jan. 15, 1938, for the ninth time, after an interval of four months. There had been no complaints of car sickness during this interval. The nine interviews had been distributed over a period of nine months (April 24, 1937, to Jan. 15, 1938). A follow-up note on March 4, 1939, one year and three months later, revealed that Edward had maintained his improvement.

The aggressive, self-assertive child and his need for self expression are illustrated by the material presented by Silvio, aged 9 years and 7 months, with an intelligence quotient of 91. This boy was the second of three brothers. The father had fits of temper, during which he would beat his wife, and finally he deserted the family when Silvio was 3 years

of age. The mother was a tense, apprehensive woman who found herself unable to cope with the financial strain of the family situation and Silvio's behavior. Her complaint was that:

He won't do what I tell him and answers me back. He picks on the other children (aged 8 and 11) all the time. He is a scary child who is afraid to stay alone. . . . He is always hitting other children. He hit a little girl the other day. He breaks bottles and upsets garbage cans. I am afraid that he will hurt somebody. He is sent home from school nearly every other day. I am a nervous wreck from him. He gets on my nerves. When I tell him to do something, he won't do it.

Direct conversation (in a previous interview) had resulted in the following record:

Q.—How are you? *A.*—All right.

Q.—Why did you come to the hospital? *A.*—For being restless in school.

Q.—How do you like your teacher? *A.*—I like her. She's nice sometimes.

Q.—Do you have to fight much? *A.*—No, Sir.

Q.—Do you like your studies? *A.*—Yes, all right.

Q.—How does your mother treat you? *A.*—All right. I'm always good at home most of the time. Sometimes I throw stones.

Since the method of direct questioning had revealed little concerning Silvio's attitudes, he was given an opportunity to express himself through the medium of several dolls representing children and adults. He was told: "Here are some boys and girls and some grown people. Let's see what is going to happen." Silvio chose a boy doll and began: "He is always bad when he wasn't supposed to be bad. He fights" (at this point Silvio banged the two boy dolls together).

Q.—Then what? *A.*—Then he goes up to his teacher and kicks her. Then he goes over to this boy and pulls his hair. Then he walks out of the schoolhouse and kicks the janitor. Then he goes home to his mother, and his mother says, "Why ain't you in school?" He says, "I been fighting. She sent me home." He kicks his mother and runs out. He sees a boy walking along the street and hits him. . . .

The physician then pointed to the dolls alternatingly as Silvio replied for each one:

Boy doll.—Why are you acting so tough?

Bad boy doll.—It's none of your business.

Girl doll.—You shouldn't show off.

Bad boy doll.—Aw, I ain't showing off. I'm tough.

Q.—The next girl says?

A. Second girl doll.—You think you're somebody, hitting everybody.

Bad boy doll.—I'm a tough guy around this block.

In a second play-interview Silvio was asked:

Q.—What did we learn here last time? *A.*—About the bad boy and the good one. This one (pointing to a boy doll) didn't do what his mother told him. He shows off most of the time. He goes around picking fights. . . . He goes around with bad boys and breaks windows of houses and hopping trucks. And he goes around tearing people's dresses. . . . And he takes his mother's eyeglasses and sets them on the ground. And he goes around hitting people with rulers. And he trips people. And he goes around at night hooking stuff and hopping trucks. And he goes around pulling people's hair. . . . And he goes around the streets and sticks people with pins. And he goes around to people saying, "You want a cracker?" and they say, "Yeh," and he cracks them in the face. And he goes around throwing sand in people's faces. When he sees dogs and cats, he throws rocks at them. He takes B-B guns and shoots out windows. And when girls have bows on their hair he pulls them off of them, and when he sees a boy with suspenders he pulls them back and hurts him. And he takes pea shooters and shoots boys. And he goes around lifting girl's dresses up. And he goes in a bathroom and pulls all the paper towels out. And he spits water all over the people. He pushes lights in and out until his mother tells him to stop. He takes burned out bulbs and throws them down the alleys. And takes two bottles and ties strings around them, and when a car goes by it will knock the bottles down and they'll break. And he takes ink and throws it on people's clean clothes. Around the house he is banging in the window and doors. He goes and pulls his mother's hair like it's a bell ringing. He takes off his shoe and hits people with it. He walks around the street and slaps kids right in the face. And he pulls people's neckties. And he scratches people's tables. And his mother tells him not to do it, (but) he fills the tub up to the top and turns the heater on full speed. He takes the screws out of the cellar doors. He goes around and pulls off girls' hats and throws them all around. He goes around and takes a hammer and bangs it on fire escapes. He yells in druggists' stores and rings doorbells and yells in there. He goes in druggists' stores and hooks something. He goes around and knocks chairs down and he breaks the legs of the chairs. . . .

Q.—Two boys are talking about this. This boy asks, "Why do you do it?" (The physician pointed from one doll to the other, the patient speaking for each doll).

Doll A.—I was just asking you.

Doll B.—You don't have to ask me nothing.

Doll A.—I don't care what you say. You ought to do what your mother says.

Doll B.—My mother is a lot of phooey.

Doll A.—You'd be sorry if you didn't have a mother.

Doll B.—My mother ain't nobody. She acts too smart. I answer back and curse her.

Q.—What is he trying to prove? *A.*—He is trying to prove that he is bad. He likes it; he feels nice and strong.

Q.—If he didn't do it? *A.*—He'd be ashamed that he couldn't fight.

Q.—What does he have to prove? *A.*—That he is tough. . . .

Why did this boy feel as he did toward his mother Silvio's attitudes concerning his mother were studied in conjunction with his relationship to his siblings. The physician placed three boy dolls about a toy table. He pointed to the smallest boy doll and then to the others, indicating that Silvio was to reply for each doll character. Silvio began by speaking for the youngest boy doll:

Youngest boy doll.—What are you always starting a fight for?

Second boy doll.—Your mother gives you more things than I get.

Oldest boy doll.—Oh, Mom don't give us more to eat than you.

Second boy doll.—Oh yes, she does too, 'cause I see her.

Q.—This boy always thinks? *A.*—That they are getting more.

Q.—All this is whose idea? *A.*—His.

Q.—What does he really think? *A.*—He thinks his mother wants to kick him out.

Q.—And? *A.*—That's why he always fights.

Q.—That shows what? *A.*—That he is scared that his mother will kick him out.

Q.—Just like? *A.*—Me.

Silvio's attitude to his aggressiveness was also studied. The physician asked:

Q.—What does he (the boy doll) want to be when he grows up? *A.*—He'd like to be a gangster and go and murder anybody. He is afraid some one will get him some day.

Q.—How does this boy feel? *A.*—He is scared that a ghost might get him in the night when he is asleep. The next time he is afraid to sleep by himself.

Q.—What else is he scared about? *A.*—That a boy might beat him up some day.

Q.—This boy is really? *A.*—Scared.

Q.—He has to show? *A.*—He is tough and bad.

Q.—Why? *A.*—He is bad (and) scared and a sissy.

Q.—The real reason that *you* try to be tough is that? *A.*—I am a sissy and afraid.

Q.—Afraid of? *A.*—A ghost might get you.

Q.—Now you can see what being tough is. *A.*—Being bad and scared and not doing what you are told. . . .

The manner in which such a fear of ghosts is handled is illustrated in the next case, which presents the problem of enuresis associated with similar fears. George was a retarded 10 year old boy with an intelligence quotient of 75. He was the third of four children of an emotionally unstable mother who had screaming and fainting spells and an easy-going father who barely made a living. He had been enuretic and "scary" since he had meningitis complicated by deafness on one side

at the age of 6. About one year previous to the first play-interview (April 21, 1938) he had been badly frightened by a boy friend who was wrapped in a sheet.

His fears were studied by placing a boy doll in a toy bed and inquiring:

Q.—What is he (the boy doll) thinking? *A.*—Something is going to get him.

Q. (another boy doll is added).—This boy says, "Who?" *A.*—A ghost is going to get him.

Q.—Then? *A.*—He wets the bed.

Q.—Why? *A.*—He is scared to get up.

Q.—And? *A.*—I don't get up to go to the bathroom.

Q.—So? *A.*—I wet the bed. I try not to be scared.

Q.—What happens? *A.*—I get scared anyhow.

Q.—Who is doing the talking? *A.*—Him, the boy.

Q.—What does he think? *A.*—The ghost is going to get him.

Q.—It's not the? *A.*—Ghost.

Q.—It's? *A.*—Him.

Q.—Who is he? *A.*—The boy.

Q.—Who is the boy? *A.*—Me.

Q.—What did you learn? *A.*—Not to be scared.

Q.—How? *A.*—That it was me thinking.

Q.—Why won't *you* be scared? *A.*—It's not a ghost. It's me thinking.

Q.—So when you want to go to the bathroom? *A.*—I'll get up and go.

Two weeks later (May 4) the mother reported:

He used to wet the bed two or three times a week. During the past two weeks he has not wet the bed once. He gets up and goes to the bathroom. He used to be afraid of the dark and now he's not. He'll go upstairs by himself now and to the bathroom. Now he goes upstairs and lights the lamp himself. (*Before?*) He was afraid. (*Why?*) I don't now. (*What made him better?*) I don't know. He used to be afraid of dead people. He isn't afraid of the dark or dead people no more. His imagination isn't as great as it used to be.

George reported:

I don't wet the bed. I wasn't scared like I was before 'cause I know it was no ghost. (*Before?*) I thought it was a ghost. (*Why?*) The boy scared me. (*Now you know?*) It's my imagination. You're thinking about it. (*Who is the ghost?*) Yourself. (*Who is that?*) Me.

The patient was seen a month later (June 11). It was learned that during this interval he had wet the bed once. George recalled that the night he wet the bed it had rained and he "got scared of the ghost."

Q.—Why? *A.*—They generally come in the rainy time.

Q.—What did you learn? *A.*—There is no ghost.

Q.—How about the rain? *A.*—I was scared.

Q.—And you thought? *A.*—That the ghost comes.

Q.—That means? *A.*—I was scared. It's me thinking.

George was seen again three months later (October 8). On this occasion his mother said:

He is coming along fine. He ain't scared no more. I sent him upstairs in the dark and he goes alone. He used to send his little brother upstairs first, then he'd go up behind him. Then when he would come down first and make his brother (aged 5) come down last. He doesn't wet the bed any more. He gets up at night and goes to the bathroom all by himself. He's real friendly with other children and he doesn't seem to be afraid of them.

George reported:

I am better. (*Why?*) There ain't no ghost. (*Why?*) I am only thinking about it.

An attempt was made at this time to get George to understand why he sent his little brother upstairs ahead of him. A large boy doll was placed beside a small boy doll:

Q.—This boy goes upstairs with his little brother. Whom does he want to go first? *A.*—His little brother.

Q.—Why? *A.*—They will grab him.

Q.—Why? *A.*—He wants the little brother grabbed.

Q.—Why? (*Boy doll.*).—Because I have to give him all my toys.

Q.—Whose idea is that? *A.*—Mine.

Q.—What did you learn today? What did you want to happen? *A.*—Him to get grabbed.

Q.—Why? *A.*—I wanted to play with my toys by myself.

George was interviewed again after an interval of six months (March 25, 1939). There had been no return of his enuresis or his fears of the dark.

The problem of timidity associated with the fear of kidnappers is presented by the next child, who was first examined in the Children's Psychiatric Clinic Nov. 21, 1935. Alice was 7 years and 5 months of age and was described as being a timid girl of average intelligence who was unable to stand up for herself. She was afraid of the dark, had alarming dreams and not infrequently referred to her fears of kidnappers. She had been carsick since the age of 3. The family always had been independent economically. Alice was the only child. The mother, who was an intelligent, emotionally stable woman, described the father as being a quiet man who "loves Alice, but he never touches her or makes over her. It's his disposition to be that way."

The patient was seen again at the age of 10 years and 3 months, after an interval of two years and 10 months (July 18, 1938). On this occasion her mother stated:

She is still shy. She has said that she doesn't like to be alone at night. She says that she is afraid that somebody will pick her up some night in a machine. She won't go anywhere alone; that is, she won't leave the block. I think it's because there is so much talk about kidnappers in the paper.

The play-interview was started by placing a girl doll in a toy bed. The physician asked:

Q.—What is she thinking about? *A.*—I believe she thinks that something will get her—you know, kidnappers.

Q.—And? *A.*—Take her away.

Q.—This girl (doll) comes up and says: Do you dream about that?

Alice spoke for each of two dolls as it was indicated by the physician:

Doll A.—Yes, I do sometimes.

Doll B.—Well, don't do that. It's never true. . . .

Q.—This girl has to learn? *A.*—Why she's afraid.

Q.—And why? *A.*—She dreams about them.

Q.—She already knows that? *A.*—It is not true.

Q.—It's? *A.*—All herself.

Q.—Why? *A.*—Because it's not true; it's make believe.

Q.—And? *A.*—She makes it herself.

Q.—Who is she? *A.*—Alice.

The next part of the play-interview was devoted to giving Alice an opportunity to understand her fear of kidnappers. A Negro male doll was placed near the toy bed. The physician then said:

Q.—Now comes the second part of the lesson. Here is a kidnapper. The girl (in bed) wakes up and sees him. *A.*—I think she'd call her mother.

Q.—Before that she looks at the kidnapper. *A.*—She is afraid.

Q.—Then the kidnapper takes off his mask. *A.*—She sees her father.

Q.—Then her girl friend comes in and says? (Another girl doll was introduced, and the physician pointed to each doll).

Doll B.—I told you not to be afraid.

Doll A.—I'll remember that.

Doll B.—Now you know it's your father.

Doll A.—Yes, I know. I know it's him and I'm not afraid.

Q.—Why? *A.*—She really loves him.

Q.—And when he comes? *A.*—She'll go with him because she loves him.

Q.—All this is? *A.*—Her dreaming.

Q.—And? *A.*—She loves her father and wants him to love her.

Q.—And? *A.*—Take her with him.

Q.—How does she do that? *A.*—She dreams that he is a kidnapper and wants to go with him.

Q.—This girl says: How about your daddy in real life? What doesn't he do? *A.*—He doesn't kiss me (and) love me.

Q.—So she dreams? *A.*—Her father is coming to love her.

Q.—What did *you* learn? *A.*—My father is the kidnapper in the dream and I think he is a kidnapper because he doesn't love me. That is why I am afraid.

Q.—All this is whose idea? *A.*—Alice's.

Q.—What is this little girl doing to herself? *A.*—She is scaring herself. . . .

Q.—You learned? *A.*—I learned that the kidnapper was my father.

Q.—Why? *A.*—He wanted to love me.

Q.—Now you won't be afraid of? *A.*—Kidnappers.

Q.—Why? *A.*—I'll know it's myself. It's what I am doing—thinking.

At this point the personal problems associated with this child's car sickness were studied as in the case of Edward. Alice was able to say: "It's my idea. . . . If I don't worry, it wouldn't happen." The physician then placed a girl doll and a mother doll in a toy streetcar. Alice was told:

Q.—Here is a girl riding with her mother. She thinks that? *A.*—If it hits anything, it will kill her mother.

Q.—Did you ever think that? *A.*—Sometimes. I think if we bumped into something, it would crash into her side and kill her.

Q.—And then? *A.*—I'd have my father to go to. Then I'd be with my father.

Q.—That's what? *A.*—I always wanted 'cause I wanted him to love me. If my mother would be struck, I'd have my father to go to.

Q.—What good is that? *A.*—I want him to love me.

Q.—It's the same as? *A.*—The kidnapper.

Q.—Suppose the mother isn't struck? *A.*—My father still loves her.

Q.—Who? *A.*—My mother, but he doesn't love the child.

Q.—So *she* thinks? *A.*—That if her mother is killed, her father would love her. If there was an accident, her mother would be killed.

Q.—All this is whose idea? *A.*—Mine.

Q.—Why? *A.*—Because *I* am thinking about it in the streetcar and in bed.

Q.—Now you can ride in the streetcar and sleep without being afraid. Why? *A.*—It's all in my own mind—what I am thinking about.

During the second play-interview, nine days later (July 27), the mother reported:

She has been fine about sleeping. She even said she wasn't afraid. She wouldn't lie on her right side on account of facing the cupboard. She'd say that something might come out of the cupboard. I can put her to bed that way now (for the past week). I sent her twice to the drugstore a block away and she didn't hurry back. She went more cheerfully and didn't say she didn't want to go alone. She still wants a light on in the living room and won't walk across the room to put on the table light. She is all right on the cars (on four or five rides since the last visit). She didn't mention a thing. (*Before?*) She'd get restless and say "I feel like I am going to get sick" when we'd get within two or three blocks of the center of the city.

Alice was able to say: "I am not afraid to go to bed by myself any more . . . It's my own idea. . . . I think of it (the kidnapping) myself." In regard to the mother's complaint about her fear of turning on the light, she could say: "I was afraid of something being in there. There's nothing in there." ("Why?") "It's my own self." Alice also reported: "I don't get sick on them (cars) any more. I think about it." ("About what?") "That it won't bump into anything." ("And?") "Kill my mother."

Q.—It's the same as? *A.*—The kidnapper. If he takes me away, I can love him.

Q.—And on the streetcar? *A.*—If there is an accident, I'll go to my father.

Q.—What did you learn? *A.*—All this is my idea.

The third play-interview occurred seven weeks later (September 10). The mother stated:

She seems better. She doesn't say anything about being afraid at all. . . . She goes into her bedroom without a light and tells me to close the door if I want to. She has been very good on the car. Before she'd take anything off the children. Now she's better. She said so herself. She said: "I don't take anything off them any more. I take up for myself." I think she has improved a lot myself. She used to cry and holler out at night. She hasn't done that for months.

Alice was given another opportunity to review the same material and was emphatic in her statement that "there wasn't such a thing as a kidnapper . . . it was my own imagination." ("What did it do?") "It made me afraid." She now could say: "I do more things. I used to be afraid. Now I can take up for myself." ("Why?") "I am not afraid of the children." Alice went on to say that the girl doll had been timid about reciting in school, and when asked why she replied for the doll character, saying: "I was afraid I'd miss and I'd get the dickens for it." At the end of this interview she could state, "It's in her (the girl doll's) mind."

Q.—And she is? *A.*—Me.

Q.—And therefore? *A.*—I shouldn't be afraid. It's my own self.

Q.—That? *A.*—Makes myself afraid.

Alice was seen for the fourth time a month later (Oct. 15) 1938). The mother could report:

I see a difference in her. She is more forward in mixing with other children. She tells me that she reads better in school and doesn't stumble over the words when the children excite her by raising their hands. She goes in the dark and doesn't say anything about it.

There had been no return of the complaint of car sickness. A follow-up visit on April 6, 1939 (six months later) revealed that Alice

had maintained her improvement. On this occasion she could speak for each doll as it was indicated by the physician and say:

Doll A.—Are you afraid any more?

Doll B.—No, I'm not afraid.

Doll A.—I'm glad you aren't.

Doll B.—I learned if I didn't think about it, I wouldn't be afraid.

Doll A.—That's good.

Q.—What did you used to think? *A. Doll B.*—I used to think a burglar would come in the window. . . .

Q.—Why did you think the burglar wanted to take you away? *A. Doll B.*—So I would be with my father.

Q.—And then? *A. Doll B.*—Us two could be together.

Doll A.—Yes, that's right.

Q.—What did you used to think about your father? *A. Doll B.*—I used to think he was mean and bad.

Q.—And that's why? *A. Doll B.*—I wanted to be with him. And I'll always think that.

Q.—Are you scared any more in the dark? *A. Doll B.*—No. I'm not afraid in the dark.

Doll A.—I'm glad you aren't. I know the dark won't hurt you.

Q.—All this was? *A.*—Myself—my own idea.

These and similar experiences have demonstrated that children can reveal their attitudes and difficulties more effectively by reenacting them in play and have led to the hope that the method of the play-interview might be of help in getting children to discuss their sex attitudes and sex knowledge without so much of the usual feeling of shame and embarrassment. After an introductory phase, during which the child learned to reply for each of a number of dolls as it was indicated by the physician, the play-interview was directed by degrees to the study of sex awareness.

The following material indicates how a 9 year old girl brought together her attitudes concerning courtship and marriage and her concept of the origin of babies. Harriet was 8 years and 3 months of age when she observed her mother dressing a chicken and remarked, "Oh, the chicken has eggs in her." Her mother explained, "This is the egg bag, and it comes down this tube and out of the hiney boots (anus)." Nothing was said about babies at this time. Four months later Harriet asked her mother where babies come from. Her mother replied, "You have to have a lot of money, and then God sends the baby down, and the mother has to get the milk and feed the baby, so she goes to the hospital." Harriet then asked: "How do poor people get so many babies?" and was given an evasive reply. When she was 8 years and 10 months of age, a girl friend told her that the baby came from a seed in the mother's stomach and not from storks.

How did this 9 year old girl synthesize this material?

The play-interview was started by placing a boy doll, a girl doll, a "mother" doll and a "father" doll before Harriet. She was asked:

Q.—What does this girl say about her boy friend?

Harriet began an imaginary conversation, speaking for each of the dolls as it was pointed to by the physician. The questions of the physician and the replies of the child were recorded verbatim by a secretary, who was in an adjoining cubicle:

Girl doll.—My boy friend is getting so much nicer, I'm going to marry him, I think, if he will let me.

Mother doll.—You're too young. Wait until next year, you'll be 18. It's up to your father.

Girl doll.—I forgot about him. He never lets me do anything.

Mother doll.—I'll go talk to him and send your boy friend in here.

Girl doll.—Sh-h-h, darling (referring to her boy friend), Mother has gone in to tell Father about getting married.

Boy doll.—That's swell.

Mother doll.—Daddy, your daughter wants to get married.

Father doll.—I'll talk to that heathen. (To girl doll) Why do you want to get married?

Girl doll.—I love him. You don't understand because you're not a girl.

Father doll.—No, wait until next year, and young man, you better keep your hands off my daughter.

The physician then spoke for the girl doll:

Q. Girl doll.—What do you mean by marriage? Why do people get married? *A. Father doll.*—To tell the truth, you better talk it over with your mother. She'll know more about it.

Harriet.—She goes to her mother.

Girl doll.—Mother, look. Why do people get married?

Mother doll.—My dear, that's a question, a real question. People get married because they want to have little babies, and you're in disgrace if you have one and are not married. If you want to get married for that reason, that's all right. If it isn't that reason, don't get married. Sometimes it's wrong to get married.

While listening to a radio program a short time previously, Harriet had heard about a baby being born to an unmarried mother.

The physician again spoke for the girl doll:

Q.—What does the baby have to do with getting married? *A. Mother doll.*—When you have a baby, the baby is the important part about you. Babies don't come from storks. We have them ourselves. They're inside of us. Understand now?

Girl doll.—Yes, Mother, but may I get married now?

Mother doll.—All right, all right. . . .

Harriet.—They dress her up for the wedding. Here's all the people. (Harriet arranges several boy dolls and girl dolls about the first girl doll and her "boy friend"). These ladies and gentlemen want to see her get married.

Q.—Why does the girl want all these people? *A.*—They're going to see her get married.

Q.—Why? *A.*—She wants to have a big wedding.

Q.—Why? *A.*—Because she's getting married and this will only come once.

Q.—So? *A.*—Here they come. The wedding is over. Here's the doctor and nurse, and she is down in the hospital. This is her mother sitting down here talking to her.

Mother doll.—See now, my dear child, you know what it is, getting married.

Girl doll.—Yes, I do, but I still like the idea. . . .

Doctor doll.—Nobody can see her now.

Girl doll.—I can't see my own husband?

Mother doll.—No, not until this is over.

Harriet.—The doctor asks the girl:

Doctor doll.—Do you know where babies come from?

Girl doll.—No, I don't. Mother has never told me.

Doctor doll.—I'll tell you because you're getting ready to have one. The baby comes from inside the woman, and a woman is made to have them. Now I think we'll take you upstairs.

Harriet.—Then she'll have a baby upstairs.

Q.—How? *A.*—I'll show you. They're upstairs, the doctor and the nurse. She is in bed and the doctor and the nurse stand around the bed. She is going to have it. Then the nurse walks in, and the doctor walks in. There are two doctors at the foot and two nurses at the sides.

Doctor doll.—The baby comes inside of you, and then out the baby comes. It's the way you want it. The baby comes from the back part of your body, and in other words, your hiney, and your baby comes from there, and from then on it's in the hospital until you're ready to go home. . . .

Q.—She would like to know how the baby got in there. *A. Doctor doll.*—That's easy. The baby is a seed inside of you, and the seed gets larger and larger, forms head, body, then arms, legs and feet, and when it's ready to come, it goes from your stomach down through a special tube and comes out the back.

Q.—She asks, "What has the father to do with the coming of the baby?" *A.*—I never heard that one.

Doctor doll.—In the first place you have to have a father to take part in the baby.

Q.—She asks, "What does that mean?" *A. Doctor doll.*—That means that the father has to take care of the baby. . . . The father takes more to it if it's a boy.

In this manner Harriet brought together what she had observed and learned at home with what she had heard from her friends and over the radio and synthesized this material into her present conception of how a baby is born. The formation of social attitudes, such as the interest in a "big" wedding and the attitude to the unmarried mother,

is illustrated in this material. The manner in which sex instruction is given to a daughter by a mother and the origin and development of this child's concept of anal birth is elicited in a matter-of-fact manner.

The child's spontaneity was encouraged through the method of the play-interview. The questions of the physician and the replies of the child concerning these sex topics were recorded verbatim and can be compared with those of 200 other children, ranging in age from 4 to 12 years. In a similar manner these children's conception of sex differences and such matters as satisfaction or dissatisfaction with the child's own sex status, sexual contents in dreams, masturbation and their reactions to the discovery of genital differences have been studied and will be reported in a later series of papers.

SUMMARY

A method has been described of working directly with the child through the medium of play-interviews. Illustrative clinical material dealing with car sickness, abnormal self assertion, parent-child relations, enuresis, fears of the dark and of kidnappers and the development of sex awareness and sex attitudes has been presented.

The procedure provides opportunities for the child to speak for each of a number of dolls. He learns to express his feelings and thoughts through the medium of these dolls as if they were responsible for all that was said and done. In this manner the child first becomes aware of and soon accepts the fact that he has contributed to his own discomfort. The question of personal responsibility and the acknowledgment by the child of the role which he plays in the total situation come up repeatedly in this method.

The clinical material seems to indicate that the attitudes, motives and imaginations of the child are closely associated with his life situation and actual experiences. Facts obtained in this manner are relatively free from adult interpretation and therefore help to contribute to a program of mental hygiene in childhood.

SEX RATIOS IN FAMILIES WITH HEMOPHILIA

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So much has been written on the subject of hemophilia that it would appear rather improbable that anything is left to be said. The present study was undertaken because of curiosity aroused by the contradictory statements of Eley [1] and of Birch [2] as to alterations in the sex ratio found in families with hemophilia. Eley has said, "As if to further perpetuate the disturbance, transmitters of hemophilia usually have more daughters than sons." Birch has stated, "The children of transmitters of hemophilia are predominantly male; 63 per cent male and 37 per cent female, or about 1.7 males to every female. . . . A transmitter has an almost 2:1 chance of having sons instead of daughters." And further on, she has said, "The children of persons with hemophilia are more apt to be female than male. . . . Nature seems to have provided that this disease shall go on forever, because the transmitters of hemophilia have more sons than daughters, while persons with hemophilia have more daughters than sons."

The usual sex ratio in most countries is about 106 to 100; that is, 106 males are born to every 100 females, and the same ratio obtains rather widely. Is it true that in families in which the disease hemophilia affects the males this sex ratio is disturbed or thrown off balance in such a way as to insure its perpetuation? Eley claimed that the transmitters have more daughters than sons, while the opposite of this was Birch's view; both, however, held that Nature strives to perpetuate the disease. Since in the majority of cases hemophilic males are destroyed by this disease before they reach the age of reproduction, most of the transmitters must come from other transmitters; that is, mothers who are transmitters produce daughters who are transmitters, so that although the persons actually affected die, the disease is carried on through a side current, as it were, of females. Thus Eley's view that transmitters have more daughters than sons would be the more logical one if Nature were striving to keep up the defect. Haldane [3] has expressed the belief

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1. Eley, R. C.: Hemophilia, Internat. Clin. **2**:202, 1936.
2. Birch, C. L.: Hemophilia, J. A. M. A. **99**:1566 (Nov. 5) 1932.
3. Haldane, J. B. S.: Blood Royal, Mod. Quart. **1**:129, 1938.

that hemophilia must constantly be arising as a mutation in stocks in which it has not occurred before; otherwise the effect of continued elimination of the affected males before reproduction would tend progressively to lower the incidence of hemophilia in the population, with its final disappearance despite the fact that females transmit it. The latter explanation would not be necessary, however, if there were a preponderance of females in the families of transmitters. The decreasing incidence expected because of the fact that the males for the most part do not reproduce would occur only if one assumes that males and females are born in equal numbers in these families.

MATERIAL

In order to test the truth of the statements as to an altered sex ratio in families with hemophilia I used the data collected in the literature. Only those families were used in the analysis in which the number and sex of all the children born in any one sibship were given. Thus if, as often happens in the records, the statement is made that there were 4 males, 4 females and 5 other children (with no statement as to sex) who died in early infancy, the record was discarded. Only when the record was complete, with the sex of all the children being given, was it included. Of course, if the records for earlier generations were not complete, or if those for some of the sibships were not fully detailed, that did not prevent the utilization of the information on the later generations of the pedigree or on the sibships on which the records were all that was desired. All those records in which the information was of the following character were likewise discarded. "A bleeder had three bleeder brothers, two bleeder nephews by two sisters, two bleeder maternal uncles." Since here, apparently, only the affected males and only enough of the transmitter females to permit one to trace the obvious descent of the defect are listed, the record was treated as incomplete unless it was definitely stated to be complete. The most extensive records were, of course, those of Bulloch and Fildes,[4] who collected all the cases up to 1911; Schloessmann,[5] who reported some 20 families; Birch,[6] who reported a large number, and Muir,[7] who compiled an extensive pedigree in a South African family.

ANALYSIS OF PEDIGREES: TRANSMITTERS

It is true, although at first not obvious, that if one wishes to determine whether there is an excess of males or of females in the families of transmitters one cannot determine the percentage of males or females in excess of the average by comparing the total number of males to the

4. Bulloch, W., and Fildes, R.: Hemophilia, University of London, Francis Galton Laboratory for National Eugenics, Eugenics Laboratory Memoirs 12 and 14, 1911.

5. Schloessmann, H.: Die Hemophilie in Württemberg, Arch. f. Rassen- u. Gesellsch.-Biol. **16**:29, 129, 276 and 353, 1924.

6. Birch, C. L.: Hemophilia: Clinical and Genetic Aspects, Illinois Medical and Dental Monograph, Urbana, Ill., University of Illinois Press, 1937, vol. 1, no. 4.

7. Muir, J.: Heredity in Hemophilia in South Africa, J. M. A. South Africa **2**:599, 1928.

total number of females. In Birch's collection of some 18 families from American and German literature she found that 63.1 per cent of the children of transmitters were males and 36.9 per cent females. This would appear to substantiate her claim that a transmitter is almost twice as likely to have sons as daughters. In 2,211 families which I collected from the literature there were 2,713 males and 1,943 females, a percentage of males of 58.3, of females of 41.7. But does this indicate a real excess of males?

If one examines a few facts, one will be convinced that this method does not give a true picture. First, in the vast majority of cases a female must have not only males but affected males among her offspring before one can call her a transmitter. In some cases it is possible to say that a woman was a transmitter though her sons were unaffected, and these will be discussed later; but such records are relatively infrequent, by far the greater number of transmitters being recognized because they had affected sons. Therefore, to be listed as a transmitter a woman has to have sons, in most cases. Those women who were transmitters but who happened to have only daughters did not show their potentiality and so are not written into the record.

It is known that a woman who is a carrier may have the following types of children:[8] (1) boys who are normal; (2) boys who are affected with hemophilia; (3) girls who are normal, and (4) girls who are carriers. She is apt to have these in equal numbers, apart from the fact that on a large scale there are 106 boys born to every 100 girls. I shall assume for the argument that the chances are exactly equal that male or female children will result. Now, if one is to prove that a woman who has only 1 child is a transmitter, this child must be a boy, and the boy must be affected, except in rare cases. Thus, of 83 families in which transmitters had but 1 child each, in 73 the child was a boy, and in only 10 was it a girl. This does not mean that a transmitter is 7 times as likely to have male as female offspring, but it does mean that her capacity as a transmitter is much more likely to show up if the child is a boy.

x 1 family with	2 boys	both boys affected
1 family with	2 boys	both boys normal
1 family with	2 girls	both girls carriers
1 family with	2 girls	both girls normal
x 2 families with	2 boys	1 normal; 1 affected
2 families with	2 girls	1 normal; 1 a carrier
2 families with	1 boy, 1 girl	both normal
2 families with	1 boy, 1 girl	boy normal, girl carrier
x 2 families with	1 boy, 1 girl	boy affected, girl normal
x 2 families with	1 boy, 1 girl	boy affected, girl carrier

Now consider families with 2 children. What are the possibilities there? The foregoing tabulation gives a list of the possible families

8. Macklin, M. T.: Heredity in Hemophilia, Am. J. M. Sc. **175**:218, 1928.

which a woman definitely carrying a factor for hemophilia might have if she had only 2 children.

Since one cannot recognize a carrier girl until she in turn has an affected son, one can regard the girls as wholly normal as one sums up the table. It is found that in only 7 (those marked x) of the 16 possible families does the woman whom one knows to be a transmitter show her potentialities in that direction. Now analyze those seven possibilities more closely. Since there are 7 possible families of 2 children each there will be in these families a total of 14 children, of whom 10 are boys and 4 are girls. The percentage of males in families of transmitters who have only 2 children each is therefore likely to be 71.4; the percentage of females, 28.6. This does not mean that transmitters are more likely to have males than females in the ratio of 71.4 to 28.6; for one sees from the foregoing tabulation that they are as apt to have females as males. It is merely that one can identify them more easily as transmitters when they have male offspring, and hence the series studied is certain to be weighted in that direction. Before one can say that transmitters have an excess of male children, one must take all families of transmitters in which there are 2 children and determine the percentage of males and females. Unless it departs widely from the expected 71.4 per cent, one can feel that they were producing females as often as males, but that one could not always detect that they were carriers of the defect.

Take one more family group in order to bring out the next point: namely, that the percentage of boys *expected* in transmitter families is always the theoretic 50, but that the percentage *observed* deviates from that value by differing degrees depending on the size of the family. Consider the 3 child families of transmitters.

By raising the expression B + *B* + 2G to the third power one finds that there are 64 possible families of 3 children. Let B stand for the chance of having a normal boy, *B* for the chance of having an affected boy and 2G for the chances of having a normal and a carrier girl. (Since both are outwardly normal they are grouped as 2G in place of G [normal girl] and *G* [carrier girl] to simplify the expansion of the expression.) Among these 64 possible families which the transmitter mother might have there are 27 in which she would have only girls or girls and unaffected boys and so would not show that she is a carrier. There would be 37 in which she might show that she is a carrier, these will be listed now:

1 family	3 boys	3 affected
3 families	3 boys	2 affected; 1 normal
3 families	3 boys	1 affected; 2 normal
12 families	2 boys; 1 girl	1 boy affected; 1 boy normal; girl normal or carrier
6 families	2 boys; 1 girl	2 boys affected; girl normal or carrier
12 families	1 boy; 2 girls	boy affected; girls normal or carriers

In these 37 families there are 111 children; 69, or 62.1 per cent, are males, and 42, or 37.9 per cent, are girls. Here again there is an excess of males; but it will be noted that there is a closer approximation to the ideal percentage of 50 than in the case of the 2 child families. The larger the number of children in the family, the nearer does this percentage of males observed in the families approach the ideal percentage of 50. Thus it will be seen that to group all of the hemophilic families together in order to determine the percentage of males and

TABLE 1.—*Percentage of Males and Females Found in the Families of Transmitters*

1	2	3	4	5	6	7
No. of Children in Family	No. of Families	No. of Boys	No. of Girls	Percentage of Boys	Percentage of Boys Expected	Difference Between 5 and 6
1	73	73	..	100.0	100.0	
1	10	..	10			
2	115	163	67	70.8	71.4 ± 3.0	0.6
2	6	...	12			
3	132	275	121	69.4	62.1 ± 2.4	7.3
3	8	...	24			
4	105	247	173	59.0	57.7 ± 2.4	1.3
4	8	...	32			
5	94	272	198	58.0	55.2 ± 2.3	2.8
6	87	297	225	56.8	53.6 ± 2.2	3.2
7	65	266	189	59.3	52.5 ± 2.3	6.8
8	47	210	166	55.8	51.8 ± 2.6	4.0
9	37	178	155	50.1	51.2 ± 2.7	1.1
10	35	185	165	52.8	50.7 ± 2.7	2.1
11	25	153	122	55.6	50.3 ± 3.0	5.3
12	16	108	84	56.2	50.0 ± 3.6	6.2
13	16	127	81	60.7	50.0 ± 3.5	10.7
14	7	55	43	56.1	50.0 ± 5.0	6.1
16	4	37	27	57.8	50.0 ± 6.3	7.8
17	2	17	17	50.0	50.0 ± 8.6	0.0
19	2	24	14	63.0	50.0 ± 8.1	13.0
20	1	13	7	70.0	50.0 ± 11.2	20.0
24	1	14	10	58.3	50.0 ± 10.2	8.3

females is an incorrect procedure. The series must be analyzed for each group as has been done in table 1.

Here are set forth the numbers of children in the families in column 1, the numbers of such families in column 2, the numbers of boys and of girls in such families in columns 3 and 4, the percentages of boys in column 5 and the percentages of boys expected in such families, with the standard deviations, in column 6. The differences between the actual and the expected values are in column 7. In some instances there was a family in which all the children were girls, and the fact that the mother was a transmitter was indicated by the occurrence of hemophilia in the sons of 2 or more of these daughters. There should be as many families in which there were all girls as there are families in which there were all boys, but it is obvious from the records that

there are not. Therefore, since the expected percentage of boys in such families was worked out omitting all families with only girls in them, these all-girl families are omitted from the final calculations in table 1 and are indicated in bold face in each case to make the record complete. In 2 child families the observed value was 70.8 per cent males; the expected value was 71.4 ± 3. Thus the apparent excess of boys was not really as great as the excess one expected to find, being actually 0.6 per cent lower. But as this difference is only a fifth of the standard deviation, one confidently says that there is no reason to suppose that this departure from the expected value was indicative of Nature's striving to have more or fewer boys than usually are found in a series of families.

One must remember, however, that in large groups 106 boys are born to every 100 girls; therefore, instead of the ratio of 50:50 which was used in the analysis to determine the ratio of boys to girls expected in these families, one should use 51.5:48.5. Thus there would be 51.5 normal boys born to every 51.5 hemophilic boys born to every 97 girls born in these families. This ratio has been used in only the 2 and 3 child sibships, as it makes a great deal of calculation and does not alter the figures greatly. Thus, instead of the 71.4 per cent of boys expected in 2 child families, there should be 71.7 per cent, and the observed difference is 0.9 per cent instead of 0.6. The standard deviation is 3, and again the difference is less than a third of this value.

Using this more complex ratio of 51.5 boys born to every 48.5 girls in 3 child families brings the expected percentage up to 63.1 instead of 62.1. The difference between the observed and the expected value becomes less in this case and is 6.3, which is only about 2½ times the standard deviation.

The use of the ratio of 51.5 males to every 48.5 females would in all cases bring the expected ratio somewhat higher than the one listed in column 6 of table 1, until at the end it would be 51.5 in place of 50. As a matter of fact, the ratio expected theoretically never quite reaches 50, but it approaches so closely to 50 that this value has been used. With 51.5, also, the expected ratio would always be a little higher, but again would approach 51.5 as a limit and would come so close to it as to be negligible. Even using the lower ratio of males, namely, 50, in place of 51.5, brings the expected and the observed value so close together as to make it as reasonable to assume that the increased percentage of males is due to the sex-linked character of the disease as to interpret it as an attempt on the part of Nature to increase the number of males. There are more males, it is true, but the excess number agrees so closely with the *expected excess* that one is justified in the interpretation that in the families of transmitters there is no greater tendency to the production of males than there is in the families of other women, and that

a transmitter is not more apt to have sons than daughters or daughters than sons.

There is a way in which one can test this conclusion. The majority of transmitters have been used because they had sons who bled. If one could find another criterion in the choice of transmitters, might one find that they had as many girls as boys? It is known that all daughters of hemophilic males are transmitters, since each girl receives the affected sex chromosome from her father. One also can find transmitters carrying this trait latent whom one knows not by the fact that they have affected sons but because in each instance a number of the daughters had affected sons. It is true that a woman may or may not have had sons herself and that the sons may or may not have been bleeders; but the final criterion by which we judge her to be a transmitter is that 2 or more of her daughters transmitted the disease. Also, if a woman had a brother who was a bleeder she may be included though only 1 of her daughters transmitted the disease. One may also call a woman a transmitter if a maternal uncle was a bleeder and 1 daughter had an affected son; or if she had a sister and a daughter who were transmitters. One now can see what are the sex ratios in the families of this type of transmitter.

Using the pedigrees that I collected, I found that there were 1,760 children of such transmitters, judged by some criterion other than that they had sons who bled. Some of them did have such sons; many others did not. Of this total, 870 were males and 890 females. This makes a percentage of 50.6 per cent females and 49.4 per cent males, when the expected percentage would be 50 per cent $\pm$ 1.2. But here again it will be noted that a great degree of selectivity was used. In many cases the transmitter had to have at least 1 daughter and usually 2 before she could be proved a transmitter; therefore, one would expect an undue number of females among the offspring of transmitters chosen in this way. I therefore separated these selected transmitters further into two classes: (1) those who were the daughters of bleeders and were chosen on that fact alone and not because of any character in their offspring, and (2) those transmitters who were not daughters of bleeders and who were selected because their daughters had hemophilic sons.

The transmitters who were daughters of bleeders had a total of 570 children, of whom 329, or 57.7 per cent, were males and 241, or 42.3 per cent, were females. Here there is an expected percentage of 50 $\pm$ 2.1. Although the difference is more than three times its standard deviation, one might well hesitate to assume that this type of transmitter has a tendency to produce more males than females. If one had had another group of 570 children, selected in the same way as this, one might find that its value deviated from the value of 57.7 by as much

as 8.8 (2×4.4) yet both groups be representative of series in which one expected 50 per cent as the ideal value. It would therefore appear that the departure from the expected sex ratio in these families may be interpreted as not being other than what one would expect in such a sample from the population of transmitters' families.

The members of the other group were selected because of their daughters' offspring, and in table 2 are found the number of offspring, the percentage of males and females among them and the expected percentage of females; for here one can calculate again what percentage of females one would expect to find in these families. For the most part

TABLE 2.—*Sex Ratio in the Families of Transmitters Other Than Daughters of Bleeders*

1	2	3	4	5	6	7
No. in Family	Total No. of Children	Males	Females	Percentage of Females	Percentage of Females Expected	Difference Between 5 and 6
1	9	0	9	100	100	
2	22	7	15	68.1	71.4 ± 9.6	3.3
3	102	36	66	64.7	62.1 ± 4.8	2.6
4	100	29	71	71	57.7 ± 5.0	13.3
5	95	38	57	60	55.2 ± 5.1	4.8
6	168	78	90	53.5	53.6 ± 3.8	0.1
7	105	44	61	58	52.5 ± 4.9	5.5
8	104	52	52	50	51.8 ± 4.9	1.8
9	117	52	65	55.5	51.2 ± 4.6	4.3
10	90	44	46	51	50.7 ± 5.3	0.3
11	121	73	48	40	50.3 ± 4.5	10.3
12	24	12	12	50	50.0 ±10.2	0.0
13	78	44	34	36.2	50.0 ± 5.7	13.8
14	14	7	7	50	50.0 ±13.3	0.0
16	48	30	18	37.5	50.0 ± 7.2	12.5
17	17	9	8	47	50.0 ±12.1	3.0
20	20	12	8	40	50.0 ±11.2	10.0
Total	1,234	567	667			

one sees that although there is a preponderance of females, namely, 667, or 54 per cent, this preponderance is what one would expect on the basis of one's mode of selecting these transmitters.

I have not proved that there are not more boys than girls in the families of carriers or that these women are not more liable to have boys than girls, but from the analysis of the data there is no reason to assume that the apparent preponderance of males in these families is due to anything other than the fact that carriers are much more easily identified when they have male offspring than when they have not.

ANALYSIS OF PEDIGREE: BLEEDERS

I now return to the statement of Birch that "the children of persons with hemophilia are more apt to be female than male." Because a bleeder is known by the fact that he bleeds rather than by some quality

which his children show, the method of analysis in this connection is not so complicated. One would expect an approximately equal number of females and males in such families. As seen in table 3, there were 130 families with a total of 525 children, of whom 240, or 45.7 per cent, were males and 285, or 54.3 per cent, were females. Here, then, is an actual excess of females when one would expect an approximately equal number of males and females. If one calculates the standard error or deviation using the expectation of equal numbers of females and males, one gets a value of ± 2.2. The difference between the expected percentage of 50 and 45.7 is 4.3, which is not quite twice the standard error and therefore may be interpreted as being due to the

TABLE 3.—*Percentage of Boys and Girls Found in Families of Hemophilic Males*

1 No. of Children in Family	2 No. of Families	3 Total No. of Children	4 No. of Boys	5 No. of Girls	6 Percentage of Boys	7 Percentage of Girls	8 Standard Deviation	9 Actual Difference*
1	24	24	9	15	37.5	62.5	10.2	12.5
2	33	66	35	31	53.0	47.0	6.2	3.0
3	14	42	15	27	35.7	64.3	7.7	14.3
4	15	60	36	24	60.0	40.0	6.5	10.0
5	6	30	17	13	56.6	43.4	9.1	6.6
6	11	66	23	43	34.8	65.2	6.2	15.2
7	7	49	21	28	43.0	57.0	7.2	7.0
8	9	72	42	30	58.3	41.7	5.0	8.3
9	3	27	7	20	26.0	74.0	9.8	24.0
10	2	20	6	14	30.0	70.0	11.2	20.0
11	4	44	16	28	36.3	63.7	7.5	13.7
12	1	12	7	5	58.3	41.7	14.4	8.3
13	1	13	6	7	46.1	54.9	13.9	4.9
Total	130	525	240	285				

* The actual difference is the difference between the expected percentage of 50 and the actual percentage observed as found in column 6. It will be noted that in 5 instances the actual difference between the observed and the expected percentage is less than the standard deviation (in 2, 5, 7, 12 and 13 child families). In 6 instances this value is greater than the standard deviation but not twice as great (in 1, 3, 4, 8, 10 and 11 child families). In only 2 instances is the observed difference more than twice as great as the standard deviation, but it is not two and a half times as great (6 and 9 child families). In 8 instances the percentage of girls is above 50, and in 5 it is less than 50.

sample studied as easily as to a supposed departure from the usual sex ratio in the families of bleeders.

If one sets forth the families in their proper size sibships as has been done in table 3, one sees that on the whole the numbers of females and males fall well within the values one would expect if the bleeders were just as liable to produce one sex as the other. In column 1 of this table are set the numbers of children in the families; in 2, the numbers of families; in 3 the total numbers of children; in 4, the numbers of boys; in 5 the numbers of girls; in 6 and 7 the percentages of each sex, and in 8 the standard deviations from the expected ratio of 50, which is the same in all families here, in contrast to table 1, where

the value differed for each size sibship. The actual differences between the observed and the expected percentages of males are listed in column 9, where they can be compared with the standard errors in column 8. Using the value of 51.5 per cent males and 48.5 per cent females does not alter the value of the standard error sufficiently to invalidate the results. It may be seen that in 8 instances the percentage of females was more than 50 and in 5 less than 50. In 5 instances the difference between the number of males (or females) and the expected 50 per cent is *less* than the standard deviation. In 6 instances this value is greater than the standard deviation but less than twice this value. In only 2 instances is the difference greater than twice the standard deviation, and even here the difference is only 2.4 times as great.

One might sum up the findings, then, in this way. It is true that there are more females than males in the children of this particular group of bleeders. This is true in the different size sibships more often than it is not true. But all of the values here recorded can just as correctly be interpreted as due to the deviation of this set of families from the standard expectation of equal numbers of males and females as it can be interpreted on the basis that the bleeders tend to have an undue number of females so as to perpetuate the disease. Inasmuch as the determination of sex is accomplished at fertilization, and inasmuch as the sex of children already born can scarcely influence the sex of children yet to be born, it is more reasonable to interpret the aforementioned findings as a variation of the normal sex ratio which obtains in this particular sample than to explain them as an attempt to break that law of sex determination.

RATIO OF UNAFFECTED TO AFFECTED MALES

Birch made the statement from her observations on the families she has studied that "the sons of transmitters have a 2:1 chance of having hemophilia." Earlier in this paper I have made the statement that the sons of a transmitter have an equal chance of having the disease or of being normal. Thus 50 per cent of sons of transmitters should be normal. This statement holds, of course, only if *all* the transmitters are recognized and if their normal sons as well as their affected sons are counted. Because one finds the transmitters more easily when they have affected sons, one would expect to find the percentage of affected males in any series higher than 50 per cent, because one will have omitted all the normal males of women who failed to have affected sons. Thus if a woman has but 1 son, he must be affected if the family is to count in the series; therefore, in 1 male families, 100 per cent in place of 50 will be affected. In families with but 2 males, one sees, if one goes back to the tabulation on page 1217, that there are only 3 families

with 2 boys; and of these 6 boys, 4 are affected, 2 normal. Therefore in families in which there are only 2 boys one expects to find 66.6 per cent affected in place of 50. The percentage of affected boys which one expects will differ for the different size sibships and must be calculated separately for each size family. This has been done in table 4. One finds that there is a total of 2,629 males in the families studied and that, of these, 1,626, or 61.8 per cent, were affected. The percentage of males which should be affected was calculated, and the sum was 1,528, or 58.1 per cent ± 0.97. The difference between 61.8 and 58.1 is 3.7, which is 3.8 times as much as the standard error. One might be inclined to interpret this as actually confirming the idea of Birch that the males in these families are more apt to be affected than unaffected.

TABLE 4.—*Ratio of Affected to Unaffected Males in Families with Hemophilia*

1	2	3	4	5	6	7	8
No. of Males in Family	No. of Families	Total No. of Males	Total No. of Affected Males	No. of Affected Males Expected	Percentage of Males Affected	Percentage of Males Expected to Be Affected	Difference Between Columns 6 and 7
1	191	191	191	191	100	100	0.0
2	195	390	287	260	73.5	66.6 ± 2.4	6.9
3	144	432	290	246	67	51.1 ± 2.4	9.9
4	109	436	254	232	58.2	53.3 ± 2.4	4.9
5	77	385	226	199	58.6	51.6 ± 2.5	7.0
6	47	282	149	143	52.6	50.8 ± 3.0	1.8
7	25	175	89	88	50.8	50.4 ± 3.8	0.4
8	17	136	69	68	50.7	50.1 ± 4.3	0.6
9	7	63	28	31.5	44	50.0 ± 6.3	5.6
10	2	20	5	10	25	50.0 ± 11.1	25.0
11	4	44	14	22	32	50.0 ± 7.5	18.0
12	3	36	11	18	30.5	50.0 ± 8.3	19.5
13	3	39	13	19.5	33.3	50.0 ± 8.0	16.7
		2,629	1,626 61.8%	1,528 58.1%			

There is one point to be remembered, however, and that is this: In the smaller size families, those of 1, 2 and 3 boys, many such families are incomplete, and only those with affected males have been selected. For example, if a woman had 2 sons at the time the record was made, and if she were going to have 4 sons before her family was complete, her family would not be included in the record if her first 2 sons were normal, the last 2 sons being the ones who were to be the affected males. The family would be included if the first 2 were affected and the last 2 were to be the normal males. Therefore, in the smaller families there is a preponderance of affected males. In the families which have 6 sons and more one sees that the percentage of those affected agrees fairly well with the percentage of affected which is expected, except in the cases in which the families are very few. But just as one could determine the sex ratio in the families of transmitters apart from any

adjustment of the percentage by using only those transmitters who were recognized by the fact that their fathers were hemophilic males, so one can use the offspring of these same women for the determination of the percentage of affected and unaffected males which one should obtain. Here one will not have to choose families with affected males to begin with; one can select the families purely on the basis that the mother had a hemophilic father. Thus one will find some women who have no sons at all, some who have sons who are unaffected and finally women who have affected sons. These data were gathered from the records used for the rest of this study, and I found that there was a total of 359 males who were sons of transmitters, the latter being identified because their fathers were bleeders. (It will be noted that this total is a little more than the total of males mentioned before in the determination of sex ratios in these families. This was due to the fact that in 7 families the total number of males and the number of affected males were given, but the statement as to the number of daughters was not complete. These 7 families could therefore be used in this determination although not for the sex ratio determination.) Of these 359 males, there were 191, or 53.2 per cent, who were normal and 168, or 46.8, who were affected. The expectation was 50 per cent $\pm$ 2.2. Therefore, the difference between the observed and expected values for the affected percentage is only 3.2, which is less than twice the standard deviation. Therefore one may interpret these results as in agreement with the theoretic explanation of inheritance of hemophilia, namely, that 50 per cent of males whose mothers are transmitters are apt to be affected. The excess of affected males in such families collected from the literature is most probably due to the degree of selectivity which one must use in identifying the transmitter, which is that she must have, in order to be identified, the very type of son whose incidence one is attempting to determine.

SUMMARY

The sex ratios obtained in the children of women who are carriers of hemophilia and in the children of hemophiliac males have been examined to determine whether the apparent departure from normal is indicative of a disturbed sex ratio in these families or whether it is caused by the method of selecting material for study.

It was found that in families of transmitters collected at random there are more males than females in a ratio of 58.3 to 41.7. This was found to be due to the fact that most transmitters are recognized only because they have males in their families in whom the disease develops. When the data are analyzed computing the number of males one should expect, the excess is found to be an apparent, not a real one.

In the families of hemophilic males in which daughters have been said to predominate, there was a slight excess of females in a ratio of 54.3 to 45.7. Here again the departure from the expected value was not outside the limits of what might occur in a sample of this sort.

It has been claimed that the ratio of affected to unaffected was much higher than the expected 50:50 ratio. Again, because of the fact that a woman is known for the most part as a transmitter because she has affected sons, the material used to determine the incidence of affected males is weighted in favor of the affected group at the outset. If the incidence is determined only from families of transmitters who are identified by the fact that they had hemophilic fathers, this preponderance of affected males disappears, and the ratio of affected to normal is well within the limits of the expected 50:50 ratio. The unaffected actually outnumbered the affected in the series collected, namely, 53.2 per cent.

Therefore, there appears to be no adequate basis for the idea that the sex ratio is disturbed in families bearing the trait of hemophilia, males and females appearing equally in the offspring of transmitters and of affected males, and the defect appearing in about one half of the sons of women who are transmitters.

EFFECT OF HYPERIMMUNE HUMAN SERUM (LYOPHILE) AND OF SULFAPYRIDINE ON EXPERIMENTAL MURINE PERTUSSIS

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When mice are inoculated intranasally or intratracheally with saline suspensions of phase 1 Haemophilus pertussis, a definite lesion of the lungs results.[1] This type of experimental infection affords a suitable method by means of which the protective qualities of various immunizing substances may be tested.

The present status of convalescent serum and of adult immune serum in the prevention and treatment of pertussis has been described by Bradford[2] and by Meader.[3] In order to enhance the protective power of immune human serum, Jundell,[4] Kendrick[5] and McGuinness,

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This investigation has been made with the assistance of a grant from the Committee on Therapeutic Research, Council on Pharmacy and Chemistry, American Medical Association.

1. Burnet, F. M., and Timmins, C.: Experimental Infection with Haemophilus Pertussis in the Mouse by Intranasal Inoculations, Brit. J. Exper. Path. **18**:83 (April) 1937. Bradford, W. L.: Experimental Infection in the Mouse Produced by Intratracheal Inoculation with Haemophilus Pertussis, Am. J. Path. **14**:377 (May) 1938. Culotta, C. S.; Marting, F. L., and Liebow, A. A.: Whooping Cough: Observations on Experimental Infection in Mice and on Attempts at Active Immunization in Mice and in Ferrets, Yale J. Biol. & Med. **10**:233 (Jan.) 1938.

2. Bradford, W. L.: Use of Convalescent Blood in Whooping Cough, with a Review of the Literature, Am. J. Dis. Child. **50**:918 (Oct.) 1935.

3. Meader, F. M.: Convalescent Serum in Whooping Cough, J. Pediat. **14**:264 (Feb.) 1939.

4. Jundell, I.: Has Specific Serum of Adults Any Value as a Remedy Against Whooping Cough? Acta pædiat. **16**:400, 1934.

5. Kendrick, P.: A Note on the Use of Reinforced Convalescent or Hyperimmune Serum for Passive Immunization of Infants Exposed to Pertussis, J. Pediat. **9**:118 (July) 1936.

Stokes and Mudd [6] have recently suggested that the donors receive injections of vaccine prior to the withdrawal of blood. Serum prepared in this manner is referred to as hyperimmune. McGuinness and his associates preserved the serum by the lyophile process in order to prevent diminution of the antibody titer during storage. Serum preserved by this method also can be readily concentrated before injection.

Observations made on the prophylactic and therapeutic qualities of lyophile serum in the treatment of pertussis have been described.[7] In support of its preventive and its therapeutic value, it has been shown that the humoral antibodies in infants are immediately enhanced by the injection of the serum.[8]

The present report describes a study of the protective action of hyperimmune human serum (lyophile) in the murine disease. Because of the current interest in sulfapyridine, the effect of this drug alone and of the combination of sulfapyridine and immune serum is also reported.

METHOD

Three week old Swiss mice of uniform size were used. The treated and the control groups were kept in separate jars, each containing 10 animals.

In each experiment a freshly isolated strain of pertussis was used to infect the mice. The suspension for inoculation was prepared by scraping the third generation growth of the organisms from the surface of a Bordet's medium plate into an 0.85 per cent solution of sodium chloride. After thoroughly mixing, the suspension was diluted to match the turbidity of a standard, which contained approximately 10 billion organisms per cubic centimeter. The infecting dose was 0.05 cc. of a standardized suspension.

The mice were anesthetized lightly by placing them in a glass jar containing a mixture of 1 part of chloroform and 2 parts of ether. They were infected by distilling 0.05 cc. of the suspension of organisms into the nares through a short, blunt-tipped no. 26 gage needle from a Luer tuberculin syringe. Preliminary testing by this method of the virulence of freshly isolated strains for mice showed that the resulting mortality was fairly uniform and varied between 80 and 100 per cent, with an average of 93.4 per cent in a group of twelve different strains.

As the mice died, they were brought to autopsy, and cultures were made from the lungs and from the spleen on Bordet's medium. At the end of ten days, all the surviving animals were killed for examination and culture studies.

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6. McGuinness, A. C.; Stokes, J., Jr., and Mudd, S.: The Clinical Uses of Human Serums Preserved by the Lyophile Process, J. Clin. Investigation **16**:185 (March) 1937.

7. McGuinness, A. C.; Bradford, W. L., and Armstrong, J. G.: The Production and Use of "Hyperimmune" Whooping Cough Serum, to be published. McGuinness, Stokes and Mudd.[6]

8. Katsampes, C.; McGuinness, A. C., and Bradford, W. L.: Effect of Hyperimmune Human Serum (Lyophile) on the Humoral Antibody Titer in Pertussis, this issue, p. 1234.

The effect of the therapy was measured by three criteria, viz., mortality, bacteriologic findings and pathologic findings. Each experiment consisted in the inoculation of 20 treated and 20 control mice. The testing of larger groups than this at one time was not practical. In order to compare the degrees of involvement of the lungs, a system of scoring was adopted, based on the average weight of each of the five lobes of the lung and in consideration of an estimate, by gross examination, of the amount of each lobe showing complete, partial or no consolidation. Thus, a lung in which all five lobes were completely consolidated was scored 19, while one in which no gross lesion occurred was scored 0. The arbitrary values selected describe the pathologic finding (consolidation) as follows: 0 to 5, none or slight; 6 to 14, moderate, and 15 to 19, severe or marked.

The hyperimmune serum was obtained in the lyophile state from the Philadelphia Serum Exchange, through its director, Dr. Aims C. McGuinness. It was dissolved in enough sterile distilled water to restore it to its original volume. Each mouse received intraperitoneally 0.3 cc. of the serum at the same time that it was infected intranasally and also on each of the two following days.

The sulfapyridine was supplied by Merck & Co. A 0.5 Gm. tablet was ground to powder with a mortar and pestle and suspended in 25 cc. of distilled water. Into the esophagus of the mouse 0.5 cc. (10 mg.) of this suspension was introduced from a 1 cc. syringe through a bent, dull-tipped no. 20 gage needle. Each mouse received treatments twice daily for three successive days after infection. The mice receiving the combined treatment received the full dose of serum and of sulfapyridine, as described previously.

RESULTS

The results of 4 experiments are shown in table 1. An examination of the findings indicates that, under the conditions of experiments 1 and 2, the lyophile serum definitely exerted a statistically significant[9] protection on the treated mice as measured by each of the three criteria: mortality, bacteriologic findings and pathologic findings. It also shows (experiment 3) that the combination of serum and sulfapyridine was also protective but probably not more so than serum alone. Sulfapyridine alone (experiment 4) failed to protect. This result confirms the findings of Cruickshank,[10] who observed that sulfapyridine offered no protection against this type of experimental disease.

In table 2, the results of similar experiments performed with 6 week old mice are shown. It is apparent that in these experiments, carried out before we appreciated the importance of using 3 week old mice,

9. Topley, W. W. C., and Wilson, G. S.: Principles of Bacteriology and Immunology, ed. 2, Baltimore, William Wood & Company, 1936, p. 777.

Assessment of the significance of the results was made according to the formula: Standard error of the difference in percentages $= 100\sqrt{p_o q_o \left(\frac{1}{n} + \frac{1}{n_1}\right)}$ in which p_o is the chance of death in the combined groups; q_o is the chance of survival in the combined groups; n is the number of animals in one group, and n_1 is the number of animals in the other group.

10. Cruickshank, J. C.: Sulfanilamide and M & B 693 in Experimental Pertussis in Mice, Lancet **2**:310 (Aug. 6) 1938.

TABLE 1.—*The Effect of Hyperimmune Serum (Lyophile) of a Combination of Immune Serum and Sulfapyridine and of Sulfapyridine Alone on Experimental Pertussis in Mice Three Weeks Old*

Experiment	Mortality					Bacteriologic Findings					Pathologic Findings					
						Number of Mice with Positive Cultures		Difference in Percentage of Mice with Negative Lung Cultures			Distribution of Mice According to the Degree of Involvement of the Lungs			Difference in Percentage of Mice with No or Slight Lesions of the Lungs		
	Number of Mice	Number Survived	Difference in Survival Rate, %	Standard Error, %	Ratio	Lungs	Spleen		Standard Error, %	Ratio	0-5 (None to Slight)	6-14 (Moderate)	15-19 (Severe)		Standard Error, %	Ratio
1. Serum	20	20	80	9	9:1	5	0	70	11	6:1	19	1	0	80	9.4	8.5:1
Controls	20	4				19	0				3	10	7			
2. Serum	20	12	60	11	5.4:1	15	0	25	9.7	2.5:1	7	9	4	35	10.7	3.2:1
Controls	20	0				20	0				0	11	9			
3. Serum + sulfapyridine	20	17	85	8.2	10:1	13	0	35	10.6	3:1	15	3	1	75	9.6	7.8:1
Controls	19	0				19	5				0	10	9			
4. Sulfapyridine	20	2	10	6.7	1.5:1	18	0	5	8.3	0.6:1	0	11	9	..	...	.....
Controls	20	0				19	0				0	10	10			

TABLE 2.—*The Effect of Hyperimmune Serum (Lyophil), of a Combination of Immune Serum and Sulfapyridine and of Sulfapyridine Alone on Experimental Pertussis in Mice Six Weeks Old*

Experiment	Mortality					Bacteriologic Findings					Pathologic Findings					
						Number of Mice with Positive Cultures		Difference in Percentage of Mice with Negative Lung Cultures			Distribution of Mice According to the Degree of Involvement of the Lungs			Difference in Percentage of Mice with No or Slight Lesions of the Lungs		
	Number of Mice	Number Survived	Difference in Survival Rate, %	Standard Error, %	Ratio	Lungs	Spleen		Standard Error, %	Ratio	0-5 (None to Slight)	6-14 (Moderate)	15-19 (Severe)		Standard Error, %	Ratio
5. Serum	24	20	5	12.5	0.4:1	5	1	57	12	4.7:1	18	1	5	19	13.6	1.4:1
Controls	23	18				16	3				13	3	7			
6.*Serum	20	7	30	11.7	2.5:1	18	2	10	6.7	1.5:1	2	16	2	10	6.7	1.5:1
Controls	20	1				20	3				0	20	0			
7. Serum + sulfapyridine	25	25	27	8.8	3:1	6	0	76	8.6	8.8:1	22	3	0	73	9.6	7.6:1
Controls	26	19				23	2				4	15	7			
8. Sulfapyridine	21	20	..	..	....	21	10	..	..	....	14	5	2	1	..	.....
Controls	20	20				20	13				13	4	3			

* The infecting dose was 0.05 cc. of a suspension containing 20 billion organisms per cubic centimeter.

the results of treatment were less marked. The infecting strains, moreover, though hemolytic, had been subcultured for periods varying from three to eight weeks. Thus, they may have been in the intermediate phase.

The mortality rates were very low. If we require a ratio of 3 to 1 between the difference in percentages and its standard error to indicate statistically valid results, then only in the bacteriologic findings in experiment 5 do we find that serum has exerted a beneficial influence. It is of interest, however, to note that in experiment 6, in which the mice received 0.05 cc. of a suspension of 20 billion organisms, a suggestive protection is reflected by the difference in the mortality rates of the two groups.

In experiment 7 the therapy, consisting of the use of a combination of sulfapyridine and immune serum, resulted in significant degrees of protection, as reflected by all three criteria. The data in experiment 8 confirm the results in experiment 4 and show that sulfapyridine alone, in the doses used, did not protect mice against experimental pertussis.

SUMMARY

Hyperimmune human serum (lyophile) protected young mice against death from experimentally induced infection with H. pertussis when it was injected at the same time as the organisms. Thirty-two from a group of 40 treated mice survived, while only 4 survived from a group of 40 controls. The degree of involvement of the lungs in the treated group was definitely less than in the control group. The difference between the groups in the number of mice showing negative lung cultures at autopsy was also statistically significant.

Sulfapyridine, in daily doses of 20 mg. per mouse administered for three days, failed to protect either 3 or 6 week old mice.

Combined treatment, consisting of the oral administration of sulfapyridine and the injection of immune serum, also protected both 3 and 6 week old mice, but the protection was no greater in the younger animals than that produced by serum alone.

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EFFECT OF HYPERIMMUNE HUMAN SERUM (LYOPHILE) ON THE HUMORAL ANTIBODY TITER IN PERTUSSIS

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That convalescent blood when injected into infants exposed to pertussis confers a certain degree of protection appears to be established.[1] Its value when injected after the onset of symptoms, however, has not been fully determined.

It would seem that the injection of immune serum during the catarrhal stage of pertussis would be indicated, since demonstrable antibodies do not normally appear until the height of infection occurs. Since the level of antibodies in the blood of recovered patients is relatively low, attempts have been made to increase the titer by inoculating immune adults with vaccine. Jundell[2] gave injections of freshly prepared vaccine to donors a few days prior to the withdrawal of blood. Kendrick[3] reported that the blood of hyperimmune donors was definitely protective when given to exposed infants. McGuinness, Stokes and

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From the Departments of Pediatrics and Bacteriology, the University of Rochester School of Medicine and Dentistry, Rochester, N. Y., and the Department of Pediatrics, University of Pennsylvania and the Children's Hospital of Philadelphia.

1. Bradford, W. L.: Use of Convalescent Blood in Whooping Cough, with a Review of the Literature, Am. J. Dis. Child. **50**:918 (Oct.) 1935. Meader, F. M.: Prophylaxis of Whooping Cough, ibid. **53**:760 (March) 1937; Convalescent Serum in Whooping Cough, J. Pediat. **14**:264 (Feb.) 1939.

2. Jundell, I.: Has Specific Serum of Adults Any Value as a Remedy Against Whooping Cough? Acta pædiat. **16**:400, 1934.

3. Kendrick, P.: A Note on the Use of Reinforced Convalescent or Hyperimmune Serum for Passive Immunization of Infants Exposed to Pertussis, J. Pediat. **9**:118 (July) 1936.

Mudd [4] suggested that hyperimmune human serum possesses definite prophylactic value. They also reported that 5 of a group of 15 infants seemed to be improved when treated with it after the onset of the disease.

The present report describes the results of a study of the effect of hyperimmune human serum on the titer of humoral antibodies when it was injected into infants in the early stage of the disease.

METHOD

The hyperimmune serum was made by the lyophile method [5] by Sharp and Dohme and was obtained from the Philadelphia Serum Exchange. Its method of preparation and its effect on the clinical course of pertussis have been described.[6] In brief, the serum was obtained from young adults who had had pertussis and who had received three complete courses of vaccine at intervals of four months prior to bleeding. At the time of injection the dried serum was dissolved in 10 cc. of sterile distilled water and in this way was restored to one half or less of its original volume. In this state the serum possessed an agglutinating titer of 1:1,280 to 1:5,120. These titers were of interest because they were far in excess of those usually noted in the serums of persons convalescing from pertussis and because they approached those obtained when rabbits were immunized with suspensions of Haemophilus pertussis.

In this study a group of 19 infants and 3 children (table 1) received injections of serum. Humoral antibodies were titered for 17 of the group before and after injection of the serum, which was given intramuscularly in 10 cc. amounts on successive days. The antibodies were measured by determining the opsonocytophagic reaction of the blood and by the method of slide agglutination.

The opsonocytophagic test was carried out as previously described in detail.[7] Briefly, the technic consisted of mixing 0.05 cc. of whole blood, obtained from the finger tip, with 0.05 cc. of a 1:1,000 solution of heparin in physiologic solution of sodium chloride. To this was added 0.05 cc. of a standard killed suspension of phase 1 H. pertussis. After the mixture had been incubated for thirty minutes at 37 C., smears were made, fixed and stained by the Giemsa method.

A series of twenty-five consecutively encountered polymorphonuclear leukocytes was examined, and the number of organisms engulfed in each was counted. The results were classified under three headings (table 2), which arbitrarily indicated the degree of phagocytosis as follows: none to slight, none to four organisms; definite, five to fourteen organisms, and marked, twenty or more organisms.

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4. McGuinness, A. C.; Stokes, J., Jr., and Mudd, S.: The Clinical Uses of Human Serums Preserved by the Lyophile Process, J. Clin. Investigation **16**: 185 (March) 1937.

5. Flosdorf, E. W., and Mudd, S.: Procedure and Apparatus for Preservation in "Lyophile" Form of Serum and Other Biological Substances, J. Immunol. **29**: 389 (Nov.) 1935.

6. McGuinness, A. C.; Bradford, W. L., and Armstrong, J. G.: The Production and Use of "Hyperimmune" Whooping Cough Serum, to be published. McGuinness, Stokes and Mudd.[4]

7. Bradford, W. L., and Slavin, B.: The Opsono-Cytophagic Reaction of the Blood in Pertussis, J. Clin. Investigation **16**:825 (Sept.) 1937.

TABLE 1.—*Clinical Data on Twenty-Two Patients with Pertussis Treated with Hyperimmune Human Serum (Lyophile)*

Case	Patient	Sex	Age	Week of Disease	Severity of Illness Before Treatment	Cc. of Serum Injected	Absolute Lymphocyte Count		Duration of Disease, Weeks	Apparent Therapeutic Effect of Serum
							Before Serum	After Serum		
1	C.F.	F	16 mo.	2	Moderate (pneumonia)	40	46,170	21,640	7	None
4	W.F.	M	7 yr.	3	Severe (pneumonia)	40	15,695	11,826	6	None
5	Y.B.	F	3 mo.	3	Moderate (pneumonia)	20	19,968	5,800	6	None
6	T.S.	M	11 mo.	3	Moderate (pneumonia)	20	18,560	6,600	6	Doubtful
11	S.S.	F	4 wk.	2	Critical (pneumonia)	40	8,576	9,952	4	Marked
13	R.R.	M	5 wk.	2	Critical (pneumonia)	40	16,248	10,605	5	Marked
3	T.F.	M	20 mo.	3	Moderate	40	21,280	8,965	5	Marked
8	J.S.	M	3 mo.	4	Severe (otitis media)	20	7,720	6,360	5	Marked
22	P.V.	M	6 wk.	2	Moderate	10	21,527	10,224	5	Marked
12	R.F.	M	3 mo.	2	Moderate	20	6,072	6,720	4½	Definite
2	D.V.	M	18 mo.	2	Moderate	40	31,008	23,104	4½	Marked
14	T.S.	M	5 mo.	2	Moderate (otitis media)	20	8,635	9,891	4	Definite
9	B.T.	F	4 yr.	1	Moderate (pneumonia)	20	14,850	9,360	4	Definite
10	J.B.	M	3 yr.	1	Severe (pneumonia)	40	17,280	11,960	4	Definite
17	A.S.	F	3 mo.	3	Moderate	20	14,800	12,220	5	Definite
21	F.T.	M	9 wk.	2	Severe (pneumonia)	10	12,464	7,168	4	Definite
7	F.C.	M	2 mo.	2	Mild (otitis media)	40	5,207	4,160	4	Definite
15	D.O.	M	4 wk.	1	Mild	20	12,000	8,140	2	Definite
16	M.M.	F	3 mo.	2	Mild	20	7,020	6,840	3	Definite
20	C.S.	F	4 mo.	2	Mild	10	12,371	10,254	3	Definite
18	M.L.	F	5 mo.	6	Moderate	20	14,800	3,670	8	Definite
19	G.P.	F	5 mo.	5	Moderate	20	13,500	7,480	6	Definite

TABLE 2.—*Humoral Antibody Titers of Nineteen Patients with Pertussis Treated with Hyperimmune Human Serum (Lyophile)*

Case	Patient	Cc. of Serum Injected	Opsonocytophagic Titer: Distribution of Cells According to Number of Organisms Phagocytosed						Agglutinating Titer	
			Before Serum			After Serum				
			0-4 Organisms; None to Slight	5-19 Organisms; Definite	20+ Organisms; Marked	0-4 Organisms; None to Slight	5-19 Organisms; Definite	20+ Organisms; Marked	Before Serum	After Serum
1	C.E.	40	21	3	1	3	10	12	0	20
2	D.V.	40	15	10	0	0	13	12	0	160
3	T.F.	40	11	11	3	1	3	21	0	40
4	W.F.	40	2	15	8	1	3	21	0	320
5	Y.B.	20	7	9	9	0	6	19	40	320
6	T.S.	20	2	16	7	0	10	15	0	640
7	F.C.	40	5	17	3	0	11	14	0	640
8	J.S.	20	3	15	7	0	7	18	0	40
9	B.T.	20	2	15	8	0	8	17	40	160
10	J.B.	40	3	19	3	0	5	20	0	40
11	S.S.	40	4	9	12	0	10	15	0	80
12	R.F.	20	5	16	4	2	20	3	10	80
13	R.R.	40	0	5	20	0	2	23	0	160
14	T.S.	20	0	2	23	0	5	20	5	40
15	D.O.	20	5	14	6	2	14	9	5	20
16	M.M.	20	0	15	10	3	7	15	0	20
17	A.S.	20	6	13	6	1	13	11	0	40
18	M.L.	20	5	20	0	0	9	16	0	160
19	G.P.	20	5	17	3	2	15	8	5	80
Average.....			5.3	12.7	7.0	0.8	9.0	15.2		

The slide agglutination test consisted of a modification of that employed by Griffith[8] to study streptococci. Readings were greatly facilitated by reference to the photographic charts published by Pauli and Coburn.[9]

The antigen was prepared by scraping a seventy-two hour growth of H. pertussis from the surfaces of two plates of Bordet medium into 10 cc. of an 0.85 per cent solution of sodium chloride. After the suspension was thoroughly mixed, it was filtered through a thin layer of cotton. It was then centrifuged and the packed organisms were resuspended in 1 cc. of saline solution.

Dilutions of serum were prepared in the usual manner in saline solution ranging from 1:5 to 1:1,280. One drop of the serum was then mixed with 1 drop of the suspension of organisms on the surface of a glass slide. About four such mixtures were made on the same slide. After shaking by tipping the slide back and forth with the fingers for five minutes, the final reading was taken with the naked eye. A black background below and behind and a bright light above and behind the slide facilitated the reading. Controls made up with saline solution were employed for comparison. Agglutinating titers determined by the slide method generally agreed with those obtained by the rapid agglutination method described by Kendrick.[10]

RESULTS

As revealed by table 2, there was a definite increase in the degree of phagocytosis by the polymorphonuclear leukocytes as a result of the administration of serum. In 12 of the 19 cases, the increase was marked. If we consider the average number of cells showing marked phagocytosis, we find that the value was 7.0 before the injection of serum, as compared with 15.2 afterward.

The serum of only 6 of the group showed the presence of specific agglutinins before they received serum, which is to be expected in the early stage of the disease. After the serum was received, the titers were increased in each case and ranged in value from 1:20 to 1:640, corresponding to those commonly found in convalescents. It will be noted that of the 12 infants showing the greatest increase in the opsonocytophagic power of the blood, 8 also showed the highest agglutination titers, ranging from 80 to 640.

From these data it may be safely concluded that a definite immediate increase in the phagocytic power and in the agglutinating titer of the blood occurred as a result of the injection of the hyperimmune serum.

None of the infants died, although the group included 15 under 6 months of age, an age period for which the mortality at the Rochester Municipal Hospital during the past twelve years has been 30 per cent. In

8. Griffith, F.: The Serological Classification of Streptococcus Pyogenes, J. Hyg. **34**:542 (Dec.) 1934.

9. Pauli, R. H., and Coburn, A. F.: Studies on Serological Typing of Streptococcus Hemolyticus, J. Exper. Med. **65**:595 (April) 1937.

10. Kendrick, P. L.: Rapid Agglutination Technic Applied to B. Pertussis Agglutination, Am. J. Pub. Health **23**:1310 (Dec.) 1933.

11 patients the duration of the disease was four and one-half weeks or less. It was our impression that all but 4 of the ones treated revealed some degree of improvement as a result of receiving the serum. These 22 infants are included in a group of 93 treated patients, to be reported elsewhere, among which 55 were 12 months of age or younger. There were 3 deaths in this series of 55; each of the 3 babies was moribund with bronchopneumonia when the serum was injected.

One interesting observation revealed that 15 of the 22 patients showed a significant decrease in the absolute number of lymphocytes. This finding may be signficant, since it has been suggested [11] that the lymphocytosis in pertussis results from stimulation of the hemopoietic tissues by infection with H. pertussis.

The extent of pulmonary involvement was ascertained before treatment in each case by roentgen examination. In 9 of the 20 cases, pneumonia existed before administration of the serum. In 3 of these (cases 1, 4 and 5) no clinical improvement resulted from the serum. In these cases Pneumococcus type IV, Streptococcus hemolyticus and Haemophilus influenzae, respectively, were demonstrated as secondary invaders. In no instance did pneumonia develop after the injection of the serum.

In 2 very young infants (patients 11 and 13), whose condition was critical on admission, there was marked improvement after injection of serum. In these infants none of the ordinary secondary invaders were demonstrated. It would be of particular interest if the lesions in their lungs were known to have been produced by H. pertussis since their rapid improvement could then be attributed to a specific effect of the serum. For infants 3, 8 and 22, in whom the results were also markedly beneficial, three successive mouse and plate cultures failed to reveal the presence of either pneumococci, hemolytic streptococci or H. influenzae. Since there is evidence [12] that the lesion of the lungs in pertussis is at least in part due to invasion by H. pertussis, it is probable that an immune serum of this type may represent a therapeutic measure of considerable importance. We have recently seen several striking therapeutic results in pertussis pneumonia complicated by the presence of a pneumococcus or a hemolytic streptococcus from the administration of the combination of lyophile serum and sulfapyridine. In those instances in which our cultures do not reveal their presence, we believe

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11. Tuta, J. A.: A Study of the Lymphocytosis Following Intravenous Injections into Rabbits of Suspensions and Extracts of Hemophilus Pertussis, Folia haemat. **57**:122 (March) 1937.

12. Smith, L. W.: The Pathologic Anatomy of Pertussis, with Especial Reference to Pneumonia Caused by the Pertussis Bacillus, Arch. Path. **4**:732 (Nov.) 1927.

that the injection of the serum alone is indicated, because mouse protection experiments [18] have failed to show a superiority of the combination treatment over the use of serum alone.

SUMMARY

A group of 19 infants and 3 children in the early stage of pertussis were treated by the intramuscular injection of from 10 to 40 cc. of hyperimmune human serum (lyophile).

In 19 of the group the opsonocytophagic reaction and the agglutinin titers of the blood were determined immediately before and after injection of the serum. The humoral antibodies, measured by these methods, were increased to levels comparable to those characteristic of convalescence. A definite decrease in the absolute number of the lymphocytes of the blood was noted in 15 of the 22 cases, and there was apparent improvement in the clinical course of the disease in 18.

260 Crittenden Boulevard.

13. Bradford, W. L., and Wold, M.: Effect of Hyperimmune Human Serum (Lyophile) and of Sulfapyridine on Experimental Murine Pertussis, Am. J. Dis. Child., this issue, p. 1228.

FUNCTION OF THE THYROID AND THE PITUITARY IN MONGOLISM

WITH A REPORT ON THE BLOOD GROUPS OF AMERICAN MONGOLOID DEFECTIVES AND COMMENTS ON THE DETERMINATION OF CHOLESTEROL

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In four previous publications [1] one of us (C. E. B.) has accumulated evidence that the so-called mongoloid deficiency is associated with an endocrine disorder. It has been demonstrated that the physical appearance in this condition is due to a developmental failure in growth, especially of the skull. Further studies are under way to determine how far the mental deficiency of this condition depends on the same factors.

The conception of an endocrine disorder as a primary factor differs from the opinions of most contemporary writers, who regard mongolism as a pathologic mutation due to an unknown factor in the germ cells or as an atavistic regression.

The latter view is emphasized by Down [2] and especially by Crookshank,[3] who consider mongolism as a racial regression. Our anatomic studies indicate that there is no relation between mongoloid deficiency and the Mongolian race. However, in order to approach the problem from still another angle, we studied the blood groups of 125 American mongoliod defectives. Penrose [4] was the first to show that English mongoloid imbeciles had practically the same distribution of the four blood groups as had 226 other English persons with mental

From the Research Department for the Study of Mental Deficiency, Wrentham State School.

1. Benda, C. E.: Proc. Am. A. Ment. Deficiency **43**:151, 1938; Studies in Mongolism: I. Growth and Physical Development, Arch. Neurol. & Psychiat. **41**:83 (Jan.) 1939; II. The Thyroid Gland, ibid. **41**:243 (Feb.) 1939; III. The Pituitary Body, ibid. **42**:1 (July) 1939.

2. Down, J. L.: Ethnic Classification of Idiots, London Hosp. Rep. **3**:259, 1866.

3. Crookshank, F. G.: The Mongol in Our Midst: A Study of Man and His Three Faces, ed. 3, London, Kegan Paul, French, Trubner & Co., Ltd., 1931.

4. Penrose, L. S.: Lancet **1**:394, 1932.

deficiency and 500 English soldiers and no tendency toward the distribution found for people of the Mongolian race. Our results similarly showed practically the same percentage distribution as has been reported for a total of 10,536 Americans.[5]

The frequencies of the agglutinogens A and B for our American mongoloid defectives found by Bernstein's [6] formula based on his triple allelomorph theory agree closely with those calculated for average Americans and also with the results found by Penrose for his English mongoloid imbeciles. He excluded 8 Jewish subjects from his total of 166. In table 1 we have included these 8 cases for better comparison, since it has not been feasible for us to exclude members of certain races from an American group. Table 1 also includes combined totals for the English and American mongoloid imbeciles as well as figures for Japanese and Chinese calculated from Strandskov's [5] data for comparison.

TABLE 1.—*Distribution of Blood Groups*

Subjects	Total	Number in Blood Group*				Percentage in Blood Group*				Frequencies of Agglutinogens	
		I AB	II A	III B	IV O	I AB	II A	III B	IV O	A	B
Americans (Strandskov)	10,536	477	4,121	1,208	4,730	4.5	39.1	11.5	44.9	24.9	8.3
Persons with mongolism, American	125	5	48	12	60	4.0	38.4	9.6	48.0	24.1	7.1
Persons with mongolism, English (Penrose)	166	3	83	14	66	1.8	50.0	8.4	39.8	30.6	5.2
Persons with mongolism, American and English	291	8	131	26	126	2.8	45.0	8.9	43.3	27.8	6.0
Japanese (Strandskov)	12,327	1,120	4,655	2,599	3,953	9.1	37.8	21.1	32.0	16.5	27.1
Chinese (Strandskov)	2,500	249	823	652	776	10.0	32.9	26.1	31.0	24.4	20.1

* Roman numerals indicate Moss groups and letters Landsteiner groups.

Since the frequency of agglutinogen B is relatively high in the Mongolian race and low in American and European populations, our data support those of Penrose in opposing the hypothesis of Down and Crookshank that mongoloid defect is due to racial regression.

Our clinical and anatomic studies in the development and physical features of mongoloid children revealed a definite growth disorder which suggested the need for a thorough study of the thyroid and the pituitary glands in patients with mongolism. The thyroid was of interest because the observations previously referred to suggested a reverse condition of the cretinistic disorder. A study of the pituitary was indicated since growth and development are supposed to be closely related to pituitary function and any disorder in these processes suggests itself as

5. Strandskov, H. H.: J. Immunol. **21**:261, 1931.

6. Bernstein, F.: Ztschr. f. indukt. Abstammungs- u. Vererbungsl. **37**:237, 1925. Snyder, L. H.: Am. J. Phys. Anthropol. **9**:233, 1926.

a pituitary dysfunction. The acromicry and splanchnomicry seen in mongolism as the reverse of acromegaly gave added impetus to our pituitary studies.

The histology of the thyroid and the pituitary glands in mongolism was reported in the papers previously mentioned.[1] The thyroid showed polymorphy of follicles, proliferative growth of parenchyma, lymphatic infiltration and, in young mongoloid children, marked increase in connective tissue. It was pointed out that the interpretation of these observations is difficult. Some histologic pictures seem to indicate hyperfunction, as seen in Graafe's disease. However, the thyroid was always extremely small, which is in contradiction to all experience with hyperthyroidism. The observations were interpreted as involutional changes.

In the literature there is much discussion about the function of the thyroid in mongolism. Earlier writers favored the interpretation of hypothyroidism, and many experiments with thyroid therapy were carried out. However, of late years the possibility of hyperthyroidism has attracted more interest.

In several papers Clark [7] stressed the idea that mongolism is the result of hyperthyroidism ceasing at birth, but he did not support his theory with pathologic studies. Recently C. R. Myers [8] came to the conclusion that hyperthyroidism of the mother may be an important factor in the causation of mongolism. Pennacchietti [9] reported 2 cases of mongolism in which he noticed a histologic structure of the thyroid which suggested colloid goiter. He also reported basal metabolic rates of several young patients and concluded that hyperthyroidism is present in mongolism. Talbot [10] found normal and low rates for mongoloid idiots. Fleming [11] reported on 6 mongoloid infants with normal rates for their weights. Tumpeer [12] reported a normal rate of —9 per cent for a Chinese mongoloid imbecile.

Disorder of the pituitary body has been emphasized by Timme,[13] who treated mongoloid children with pituitary extracts. He has not reported histologic studies to support his theory.

Gordon [14] reported observations on the pituitary in 2 instances of mongolism. His observations are in accordance with our own.

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7. Clark, R. M.: J. Ment. Sc. **74**:265 and 739, 1928; **75**:261, 1929; **79**:328, 1933.
8. Myers, C. R.: Proc. Am. A. Ment. Deficiency **43**:142, 1938.
9. Pennacchietti, M.: Rev. neurol. **2**:276, 1932.
10. Talbot, F. B.: Monatschr. f. Kinderh. **27**:465, 1924.
11. Fleming, G. B.: Quart. J. Med. **16**:11, 1922.
12. Tumpeer, I. H.: Mongolian Idiocy, J. A. M. A. **79**:14 (July 1) 1922.
13. Timme, W. W.: Bull. Massachusetts Dept. Ment. Dis. **14**:229, 1930.
14. Gordon, M. B.: Endocrinology **14**:1, 1930.

Raeder [15] mentioned 1 case of mongolism in which ". . . the pituitary was cystic and there was little sign of activity in the secreting cells of the anterior lobe, and marked increase in the pigmentation of the posterior lobe." In another case "an interstitial inflammation of the ovaries and hypophysis" was observed.

Our own microscopic studies in 15 cases of mongolism revealed a definite disorder of this gland. The histologic picture is characterized by a lack or deficiency of the basophilic system and an increase in eosinophilic cells, which are mostly of a small type. This was especially marked in some material obtained from children who died in the first years of life. In mongoloid patients over 6 years of age the pathologic condition of the pituitary is less obvious but still recognizable.

These observations threw some light on the obscure nature of mongoloid deficiency, which Grinker termed "a mysterious malady."

In an attempt to gain more insight into the functional state of the endocrine system in mongolism, biochemical studies should help to determine which gland has the key position in that disorder. The object of the studies reported in the present paper was to ascertain, if possible, what role the thyroid plays in mongolism. Data are presented which should elucidate this point, namely, cholesterol values and metabolic rates. As an initial biochemical study of pituitary function, several dextrose tolerance tests were made and blood sugar levels during fasting were determined.

DETERMINATIONS OF CHOLESTEROL

Methods.—The Bloor [16] method was chosen for the determination of cholesterol because of its simplicity. Our first experience, however, gave consistent results, due to precision, but quite erroneous low values. In order to rule out absolutely any possibility of loss due to transfer, to measuring, to creeping of the cholesterol film or to spattering during the evaporation in a beaker, slight changes in manipulation were introduced, which incidentally simplified the procedure. Then the method was checked by means of processed standard controls and by recovery tests.

It was found by repeated tests that when all precautions were taken a standard control treated with alcohol and ether almost invariably differed from the regular standard by as much as 16 per cent if the color developed in the dark at room temperature for ten or fifteen minutes. The color of the standards treated with alcohol and ether noticeably developed and faded more rapidly than that of the untreated standards, thus passing more quickly through the various shades of color. These observations indicated that the difficulty lay in the color development and that it was not altogether due to a brown discoloration caused by some constituent of the blood, as claimed by Mueller; [17] nor was overheating of the dry residue alone the explanation, as suggested by Bloor,[16] for when chloroform-treated standards were evaporated, heated to dryness and

15. Raeder, O. J.: Bull. Massachusetts Dept. Ment. Dis. **4**:104, 1920.

16. Bloor, W. R.: J. Biol. Chem. **24**:227, 1916.

17. Mueller, J. H.: J. Biol. Chem. **25**:549, 1916.

taken up with chloroform they checked almost exactly with unheated regular standards. Thus the effect could be due only to some sensitizing or catalyzing effect of the alcohol-ether treatment.

A difference in rate of development of color between samples and standards was noted by Bloor [18] in 1917. Reinhold [19] attributed this variability to the more rapid rate of reaction of the esters of cholesterol present in blood. He found that cholesterol palmitate, oleate and acetate reacted with the acetic anhydride–sulfuric acid mixture more quickly than did pure cholesterol. He even used these facts in a procedure for determining both the ester and the total cholesterol on the same aliquot of filtrate merely by reading the colors at different stages of development. He found that the yellow-green stage of color development was more reliable for determining total cholesterol by the usual procedures. In 1932 Mirsky and Bruger [20] stated clearly that the Liebermann-Burchard [21] reaction is not reliable if temperature, time and intensity of light are uncontrolled and that otherwise it is difficult to check results by duplicate samples within less than 10 per cent. Mühlbock and Kaufman [22] found that results with colorimetric methods differed from those with gravimetric methods by minus 46 per cent to plus 76 per cent and concluded that these large errors preclude the use of colorimetric methods for exact investigations. That the development of color has been a source of difficulty generally is evidenced by the many different directions given in the literature, by the disagreement of various methods and authors [23] and by the different and sometimes low values given for normal limits.[24]

Bloor, Pelkan and Allen [25] speed up development of color in both samples and standards to obtain the more stable yellowish green color by placing the tubes in the light of the colorimeter lamp for fifteen minutes. Schube [26] uses the yellow color developed at 20 C. in the dark for twelve to twenty-four hours. Mirsky and Bruger [20] hold the sky blue color by chilling. They obtained excellent check results by placing the cylinders five minutes in a dark closet at room temperature and then ten minutes in the refrigerator, taking them from the refrigerator one at a time for a rapid comparison of the light bluish color. In the Leiboff [27] method the tubes are chilled at once for a half minute and read after ten minutes at room temperature in the dark.

Our procedure also was to stop all reaction at the very beginning by chilling for two to three minutes the instant after addition of the reagent and then allowing sample and standard colors to develop together slowly at room tempera-

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18. Bloor, W. R.: J. Biol. Chem. **29**:437, 1917.

19. Reinhold, J. G.: J. Biol. Chem. **105**:lxxi, 1934; Proc. Soc. Exper. Biol. & Med. **32**:614, 1935; Am. J. M. Sc. **189**:302, 1935; Am. J. Clin. Path. **6**:22 and 31, 1936.

20. Mirsky, A., and Bruger, M.: J. Lab. & Clin. Med. **18**:304, 1932.

21. Grigaut, A.: Compt. rend. Soc. de biol. **68**:791, 1910. Liebermann, C.: Ber. d. d. chem. Gesellsch. **18**:1803, 1885.

22. Mühlbock, O., and Kaufman, C.: Biochem. Ztschr. **233**:222, 1931.

23. Weston, P. G.: J. Biol. Chem. **28**:383, 1917. Myers, V. C., and Wardell, E. L.: ibid. **36**:147, 1918. Mueller.[17] Mühlbock and Kaufman.[22]

24. Looney, J. M., and Childs, H. M.: Blood Cholesterol in Schizophrenia, Arch. Neurol. & Psychiat. **30**:567 (Sept.) 1933.

25. Bloor, W. R.; Pelkan, K. F., and Allen, D. M.: J. Biol. Chem. **52**:191, 1922.

26. Schube, P. G.: J. Lab. & Clin. Med. **18**:306, 1932.

27. Leiboff, S. L.: J. Biol. Chem. **61**:177, 1924.

ture in the dark for forty minutes. The colors of processed standard controls were found to be in the same stage of development as the color of the untreated standard at the end of forty minutes, and the relative development was practically the same up to twenty additional minutes in ordinary daylight. Thus the development and the fading were slowed, which allowed a longer period of equal intensity and plenty of time for careful readings without the necessity for such extreme care and speed as required by the Mirsky-Bruger technic. The colors were only a little more intense but were much less yellow and more stable and reliable than those allowed to develop thirty-two or forty minutes without chilling.

It is true that the standard in the colorimeter sometimes changed slightly if more than ten or fifteen minutes was taken for readings, but extra standards and processed controls accompanied all samples, so that a fading standard was quickly noted and replaced. Actually, unless too many determinations (more than eight) were made at the same time, the standard in the colorimeter did not fade

TABLE 2.—*Results of Cholesterol Recovery Tests* *

Direct test.....	237 250	218 224	202	163 165	217	153	180	234	163	250	309	229
Recovery test.....	254 240	225	202	169 171	212	150	180	234	163	262	324	238

* Total cholesterol, expressed as milligrams per hundred cubic centimeters of serum.

TABLE 3.—*Effect of Heating in Cholesterol Extraction* *

Mixture not heated.....	197	218 224	163 165	170	153	224 211 p	185	179	191	186	283	133
Mixture heated.....	204	214 224	163 168	170	151	231 211 p	196	179	191	188	291	130

* Total cholesterol, expressed as milligrams per hundred cubic centimeters. Serum was used except in 2 instances (p), when plasma was employed.

appreciably and still checked with duplicate standards within 2 per cent or less at the end of the readings. By our technic we obtained good results in checking the same filtrate on different occasions and made good recoveries of added cholesterol (table 2).

Since we found with Boyd[28] that heating of the alcohol-ether mixture did not increase extraction of the cholesterol, heating was omitted. A comparison of the effect of heating the mixture and the result of cold extraction is presented in table 3.

The details of the determination of total cholesterol were as follows: Two cubic centimeters of serum was added drop by drop, with shaking, to 30 cc. of an alcohol-ether mixture (3:1) in a 50 cc. volumetric flask. The volume was then raised to exactly 50 cc. with alcohol and ether, the mixture was shaken, and the flask was allowed to stand at room temperature for half an hour to twenty-four or forty-eight hours. The mixture was then filtered through Whatman no. 43 paper into a flask or large tube. A 10 cc. aliquot of the filtrate was carefully pipetted into an N. P. N. tube or, preferably, into a Folin-Wu receiving tube. Two quartz pebbles were added to aid ebullition, and the solution was evaporated

28. Boyd, E. M.: J. Biol. Chem. **114**:223, 1936.

under the hood, the tube being placed in a 1 liter enameled cup 6 inches (15 cm.) high with sufficient water to cover the level of the solution. The water was brought to boiling not too rapidly at first, while the ether bubbled off, but vigorously later, until the last traces of alcohol were removed from the sides of the tube.

In recovery tests exactly 1 cc. (or some convenient known amount) of stock standard (200 mg. of cholesterol per hundred cubic centimeters of chloroform solution) was placed in a 50 cc. volumetric flask and evaporated to dryness over boiling water. When cool the cholesterol was dissolved in 8 cc. of ether, 24 cc. of alcohol was added, and then an exact amount (1, 1.5 or 2 cc.) of serum was run in drop by drop with shaking. The procedure was then continued as for samples.

For each set of 4 or fewer samples in duplicate or with recovery tests, 10 cc. of dilute standard (8 mg. of cholesterol per hundred cubic centimeters of chloroform solution) in a Folin-Wu receiving tube with two quartz pebbles was evaporated to complete dryness and allowed to cool, and 10 cc. of alcohol-ether mixture was added. Then treatment was carried out as described for samples.

After the evaporated samples, the duplicate samples or material for recovery tests and the standard control had cooled to room temperature, exactly 10 cc. of dry chloroform was carefully pipetted into each tube. The tubes were corked and carefully rotated for mixing, and were then allowed to stand for forty minutes to an hour or longer at room temperature with occasional gentle shaking. The solutions were then filtered through Whatman no. 43 paper into test tubes. Five cubic centimeter aliquots of the filtrates were measured with clean dry pipets into either 10 cc. glass-stoppered graduates or, preferably, 15 cc. glass-stoppered centrifuge tubes in a rack. Stoppers were attached by means of elastic bands. Three 5 cc. portions of dilute untreated standard were similarly measured. With the standards staggered in the sequence, to the tubes were added in quick succession 1 cc. of a mixture of 10 parts of reagent acetic anhydride and 1 part of chemically pure concentrated sulfuric acid, which had been allowed to cool to room temperature. The tubes were quickly stoppered and inverted once, care being taken to avoid loss by popping out of the stoppers, and all were placed simultaneously in ice water and left for two to three minutes. They were then simultaneously removed to a dark place, where they were kept at room temperature for forty minutes. After the colorimeter light was adjusted by means of two of the regular standards, the third standard, the control, the samples and the recovery preparations, if any, were read.

For serums with low values more alcohol-ether filtrate was used; or, alternatively, exactly 7 cc. of chloroform was added instead of 10 cc. for the final extraction, 5 cc. being used for the development of color.

The half-hour for alcohol-ether extraction and the forty minutes for chloroform extraction were convenient but not necessarily minimal times. Boyd [28] claimed complete extraction with the alcohol-ether mixture in fifteen minutes.

The same procedure was used for oxalated whole blood, except that the blood was first added to 8 cc. of ether, 24 cc. of alcohol was then added slowly, with shaking, and the volume was finally made up to 50 cc. with the alcohol-ether mixture.

A few microanalyses were made on serum according to the Sackett [29] modification of Bloor's method.

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29. Sackett, G. E.: J. Biol. Chem. **64**:203, 1925.

A number of microtests were also made on finger blood or on oxalated whole blood according to this method, except that the blood was added directly to the ether, alcohol being added afterward, and after decantation into a Folin-Wu receiving tube, additional alcohol-ether mixture was twice added, centrifuged and decanted for complete extraction. The residue left after evaporation was allowed to soak in exactly 7 cc. of chloroform, 5 cc. of filtered chloroform extract being used for the color development. The results checked fairly well with values obtained for oxalated whole blood by the macroprocedure (table 4). However, as there seemed to be greater variations in the serum, due in part to the ester fraction, and since the blood cells contain little if any esterified cholesterol (Bloor and Knudson[30]), results for total cholesterol in whole blood alone are omitted as of less value.

Esterified cholesterol was determined by the method of Bloor and Knudson,[31] except that the alcohol-ether mixture was not heated (Boyd[32]) and except for minor changes in technic designed to avoid mechanical loss and errors in development of color. To 10 cc. or 15 cc. of the alcohol-ether-serum or alcohol-ether-blood filtrate in a Folin-Wu receiving tube (marked at 12.5 cc.) was added

TABLE 4.—*Comparison of Determination of Cholesterol by the Macromethod and by the Micromethod* *

Macromethod.....	194 s 190 s	189 s	155 b 166 s	140 b	193 b 191 b	171 b 173 b	148 b	233 b 234 b
Micromethod.....	192 s	186 s	160 b 155 b 166 b 161 b 160 s	127 b 129 f	177 b 177 f	162 b 162 f	135 f	215 f 215 f

* Values are expressed as milligrams per hundred cubic centimeters; "b" indicates oxalated blood, "s" serum and "f" finger blood.

1 cc. of a 1 per cent alcoholic suspension of digitonin. The mixture was evaporated just to dryness, 15 cc. of purified petroleum benzine was added and the tube was covered with a watch glass and heated gently in a warm water bath until the petroleum benzine had boiled down to about half its original volume. When the mixture was cool more petroleum benzine was added until the level was raised exactly to the 12.5 cc. mark. The solution was then well rotated, shaken and filtered through a fat-free cotton plug in the stem of a funnel into a test tube. A 10 cc. aliquot of the petroleum benzine filtrate was pipetted into a Folin-Wu receiving tube and cautiously evaporated to dryness, two quartz pebbles being used. When the residue was cool, exactly 7 cc. of chloroform was added. The tube was corked and allowed to stand for forty minutes or longer, with occasional shaking. Finally 5 cc. of the uniformly mixed chloroform extract was tested colorimetrically as described for total cholesterol.

Results.—With a few exceptions, the fasting total cholesterol of serums of 50 persons with mongolism aged from 2 years and 10 months to 29 years ranged from 135 to 234 mg. per hundred cubic centimeters,

30. Bloor, W. R., and Knudson, A.: J. Biol. Chem. **29**:7, 1917.
31. Bloor, W. R., and Knudson, A.: J. Biol. Chem. **27**:107, 1916.
32. Boyd, E. M.: J. Biol. Chem. **115**:37, 1936; footnote 28.

values now accepted as normal.[33] From table 5 it may be seen that 5 of the values (which happened to be those of females) were close to 250 mg., which is a high normal level.[34] One boy of 10 years had on three different occasions 312, 291 and 244 mg., respectively.

Similar values were found for 17 nonmongoloid feebleminded controls (147 to 231 mg., with a single value at 252 mg.). The consistently higher values found for 5 typical cretins were in sharp contrast (294 to 362 mg.).

TABLE 5.—*Values of Total Cholesterol of Serum During Fasting*

Persons With Mongolism						Controls					
Male			Female			Male			Female		
Age		Cholesterol, Mg./100 Cc.	Age		Cholesterol, Mg./100 Cc.	Age		Cholesterol, Mg./100 Cc.	Age		Cholesterol, Mg./100 Cc.
Yr.	Mo.		Yr.	Mo.		Yr.	Mo.		Yr.	Mo.	
2	10	227	5	0	200	4	4	216	6	7	228
7	1	140	6	1	245	7	1	210	6	10	199
7	6	180	6	7	219	9	4	252	12	4	226
7	9	188	7	1	179	9	8	196	12	5	231
8	0	247	7	5	215	11	6	157	14	7	181
8	2	163	8	6	220	14	1	174	14	8	229
9	8	189	9	2	234	16	3	147	14	10	185
10	4	198	10	0	170	24	1	163	14	10	211
10	4	312	11	3	248				17	0	231
10	7	291	11	4	152						
10	9	244	11	5	170						
10	10	144	11	6	178						
11	2	205	11	6	168						
12	2	223	13	2	228						
12	3	170	14	0	197						
12	3	202	14	1	224						
13	5	191	15	8	248						
13	9	173	16	5	236						
14	6	206	17	7	163						
14	7	149	17	11	135						
15	8	151	18	1	140						
16	11	150	20	3	217						
17	3	181	25	3	268						
17	7	201	26	0	254						
18	5	192	27	8	248						
19	5	196	28	5	254						
22	6	155	29	0	234						
23	3	207									
23	10	140									
27	9	194									

Cretins					
Male			Female		
Age		Cholesterol, Mg./100 Cc.	Age		Cholesterol, Mg./100 Cc.
Yr.	Mo.		Yr.	Mo.	
22	3	262	15	10	294
23	5	317	16	7	294
			31	7	354
			32	3	359
			33	0	362
			41	9	357
			42	7	335

The normal cholesterol levels of patients with mongolism indicate that this affliction, at least after the age of 2 years, is not essentially a condition of either hypothyroidism or hyperthyroidism. The values are consistent with a hypopituitary state, as Hurxthal and Hunt[33b] found the same proportion of high normal and normal cholesterol values (2 and 12, respectively) in 14 cases of hypopituitarism.

33. (*a*) Offenkrantz, F. M., and Karshan, M.: Serum Cholesterol Values for Children, Am. J. Dis. Child. **52**:784 (Oct.) 1936. (*b*) Hurxthal, L. M., and Hunt, H. M.: Ann. Int. Med. **9**:717, 1935. (*c*) Looney and Childs.[24]

34. (*a*) Page, I.; Kirk, E.; Lewis, W.; Thompson, W., and Van Slyke, D. D.: J. Biol. Chem. **111**:613, 1935. (*b*) Sperry, W. M.: ibid. **114**:125, 1936.

Results of a few tests which are presented in table 6 show the same relation as the values for the controls between the total cholesterol and the cholesterol esters of whole blood and of serum. It is seen that the values for total cholesterol in the whole blood and in the serum are not always so nearly equal and interchangeable as might be inferred from the literature.[35] The total cholesterol in the blood tends to be nearer or equal to the total in the serum when it is low. The percentages of ester in mongoloid serum, varying from 61 to 80, were about the same as those of 5 feebleminded controls, 57 to 71. These results correspond with published data for normal plasma or serum.[36]

DETERMINATION OF BASAL METABOLIC RATE

Method.—The basal metabolism tests were made with a carefully checked Sanborn motor-grafic apparatus. From two to five tests of at least two eight minute periods were made on each patient. The first test was devoted primarily to acquainting the child with the apparatus and with the procedure. In most of the cases close checks on different days were finally obtained, the lower value being chosen.

The usual preparations for the test were made, breakfast being withheld and great care taken to have the patients comfortable and relaxed. It was necessary to do the testing fairly early in the morning, before the children should become restless. General conditions were noticed such as playfulness, sleepiness, nervousness, tenseness and sweating. Emphasis was placed on simplicity of procedure and rapport between operator and patient.

The children were remarkably cooperative, probably because the tests were made at their dormitories amid the usual surroundings. The comforting presence of the attendant or matron was helpful. Both extreme quiet and extreme noise were avoided, the whole performance being rather casual. Often a playmate would be allowed to sit in the room quietly. Sometimes the children were best initiated together, two or three first tests being made successively in the same room on the same morning.

Results.—When based on the Mayo Foundation standards[37] the basal metabolic rates of 25 persons with mongolism are on the minus side (table 7). None are plus, 8 are between 0 and —10 per cent, 8 range from —11 per cent through —15 per cent and 9 are below —15 per cent. The calculations based on the Talbot standards[38] for children give different results. With use of the weight standards, 4 of the rates are on the plus side, and only 4 are below —15 per cent

35. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1931, vol. 1.

36. Boyd, E. M.: J. Biol. Chem. **110**:61, 1935. Klein, W.: Ztschr. f. physiol. Chem. **92**:302, 1914. Mühlbock and Kaufman.[22] Bloor and Knudson, footnotes 30 and 31. Offenkrantz and Karshan.[33a] Page and others.[34a] Sperry.[34b]

37. Boothby, W. M.; Berkson, J., and Dunn, H. L.: Am. J. Physiol. **116**:468, 1936.

38. Talbot, F. B.: Basal Metabolism Standards for Children, Am. J. Dis. Child. **55**:455 (March) 1938.

(excluding subject 24, an adult). When the Talbot height standards are used, all rates except 1 (— 17 per cent) come into normal range. In contrast, all calculations for 3 typical cretins, interpolated because of the patients' small stature, were below normal limits, whether based

TABLE 6.—*Total and Esterified Cholesterol Values of Serum and of Whole Blood*

Subject	Sex	Age Yr.	Age Mo.	Cells, %	Total Serum Cholesterol, Mg.	Total Blood Cholesterol, Mg.	Serum Ester Mg.	Serum Ester %	Blood Ester, Mg.
					Persons with Mongolism				
1	M	2	10	..	227	...	144	62	..
					227	...	137	..	..
2	M	7	9	41	188	171	124	68	69
					188	170	129	..	68
					186	...	128	..	..
3	M	10	7	46	291	233	206	70	84
					283	234	194	..	81
		10	9	46	244	200	196	80	114
						201	...	..	107
4	M	12	2	..	218	193	...	..	..
					227	191	...	..	..
5	M	13	5	45	189	165	116	61	56
					191	165	116	..	59
					191	...	109	..	..
6	F	17	11	40	130	145	88	66	53
					133	148	88	..	55
						149	...	..	..
		18	1	41	140	141	103	74	56
					140	140	103	..	..
7	M	18	5	..	195	164	...	..	..
					189	169	...	..	..
8	M	23	10	...	140	140	...	..	..
					140	140	...	..	..
9	F	29	..	..	233	191	...	..	..
					234	199	...	..	..
					Controls				
10	M	4	2	..	216	...	138	63	..
							131	..	..
11	F	7	7	44	231	182	161	71	101
					224	184	161	..	104
12	M	11	11	..	...	179*	...	..	..
		12	2	..	179	...	100	57	..
					179	...	104	..	..
13	M	14	1	..	178	171	...	..	..
					174	173	...	..	..
14	F	18	5	..	240	201	...	..	..
					236	201	...	..	..
		18	5	..	273	202*	...	..	..
					275	200*	...	..	..
		18	5	..	246	209*	...	..	..
					253	...	...	..	..
15	F	Adult		..	199	...	125	63	..
16	F	Adult		35	185	183	127	69	88
						184	129	..	82

* Determined by micromethod.

on Mayo Foundation standards, on Talbot weight and Talbot height standards (although 2 subjects were adults) or on Harris-Benedict standards. Complete data are given for reference. It is of interest that the accompanying cholesterol values, not necessarily for the same day as the metabolic rates, show no relation to metabolic rate for the mongoloid subjects.

TABLE 7.—*Basal Metabolic Rates of Persons with Mongolism, Cretins and Controls* *

Subject	Sex	Age Yr.	Age Mo.	Height, In.	Weight, Lb.	O_2, Cc. per Min.	Cal. per 24 Hr.	Metabolic Rate, % Mayo	Talbot Wt.	Talbot Ht.	No. of Tests	Cholesterol, Mg. per 100 Cc. Serum
Persons with Mongolism												
1	F	6	1	40½	39	98	681	—22	—14	—10	2	219
2	F	6	1	40	33	107	744	— 8	+ 4	0	2	245
3	M	6	8	42	38½	122	...	— 9				
		6	11	42¼	38	121	841	— 9	+ 7	+ 3	4	140
4	F	7	0	43½	47	121	...	—12				
		7	0	43½	47	120	834	—13	— 5	+ 3	2	215
5	F	7	1	41¼	38½	105	730	—14	— 7	— 5	4	179
6	M	7	6	47½	55	155	1,077	— 8	+ 5	+14	5	180
7	M	7	9	44¼	43	119	827	—18	— 3	— 2	2	188
8	M	8	1	44½	46	127	...	—13				
		8	2	44½	46	128	890	—14	— 1	+ 4	3	247
9	M	8	2	46	52	128	...	—18				
		8	2	46	52	119	827	—24	—11	— 5	2	163
10	F	8	6	42	41	93	646	—24	—21	—17		
		8	6	42	40½	99	688	—18	—15	—12	2	220
11	F	9	1	44¼	43½	110	764	—13	—10	— 8		
		9	1	44¼	43½	113	...	—10	....	....	2	234
12	F	9	0	42	46	112	778	—10	—11	— 1		
		9	7	42	46	116	...	— 7				
		9	7	42	46	115	...	— 8	....	....	4	170
13	M	10	2	48½	60	149	...	— 9				
		10	2	48½	60	140	973	—14	—11	+ 1	2	189
14	M	10	3	49¼	58	134	...	—20				
		10	3	49¼	59	128	899	—22	—18	—10	2	198
15	M	10	7	52	65½	147	...	—16				
		10	7	52	66	144	1,000	—18	—12	— 6	2	312
16	F	10	9	50¼	58	133	924	—15	— 7	— 4	2	248
17	F	10	11	50¾	61	155	...	— 4				
		10	11	50¾	62	156	...	— 4				
		11	2	51¾	66	161	1,119	— 4	+ 5	+11	4	152
18	F	11	0	52½	79	154	...	—15				
		11	0	52½	80	143	994	—22	—16	— 3	2	168
19	M	11	1	48½	58	135	938	—15	—13	— 3	3	205
20	F	11	6	49	56	119	827	—21	—15	—12	2	178
21	M	12	3	51	71	147	1,022	—16	—14	— 2	5	202
22	M	13	5	52¼	63	155	...	— 8				
		13	6	52¼	64	151	1,049	—11	— 6	— 3	3	191
23	F	18	0	54	104	151	...	— 9				
		18	3	54	102	150	1,042	— 9	—22	— 4	3	140
24	F	26	2	54½	101½	157	1,091	— 2	(—18)	(— 1)	3	268; 254
25	F	29	5	59½	100½	164	1,139	— 4	(—14)	(—13)	4	234
Cretins												
26	F	16	7	46½	58	95	660	—23	—33	—25	2	294
27	F	33	1	51	88½	119	827	—17 (Harris-Benedict, —26)	(—33)	(—16)	2	362
28	F	42	8	55	150½	134	961	—30 (Harris-Benedict, —31)	(—44)	(—18)	2	335
Controls												
29	F	5	4	40¾	34	92	639	—23	—12	—16	2	210
30	F	6	7	45¼	46	141	979	— 1	+12	+16	2	228
31	F	6	8	47	56	145	1,008	—10	+ 4	+14	2	200
32	M	6	10	48¼	63	154	1,070	—15	— 4	+11	2	210
33	M	8	3	50½	47	140	972	—11	+ 8	— 5	2	182
34	M	8	9	47	39	129	896	— 9	+12	— 3	2	
35	M	9	5	52	59	164	1,139	— 6	+ 5	+ 7	2	196
36	M	11	3	55½	70½	180	1,251	— 3	+ 5	+ 6	2	157
37	F	12	3	55	85¼	200	1,390	+ 6	+14	+23	2	226
38	F	14	9	57	79	153	1,063	—11	— 9	—12	2	185
39	F	15	8	59	83½	203	1,410	+17	+17	+ 9		
		15	8	59	84	208	1,445	+20	+20	+12	3	181
40	F	17	0	61¾	128	176	1,223	—15	—19	—14	2	231

* Parentheses are used around the Talbot figures when the patients were adults, because the Talbot standards are for children.

Certainly our normal or low results rule out hyperthyroidism as a common finding in cases of mongolism. We observe that the 6 metabolic rates reported by Pennacchietti [9] also do not support his view that mongolism is a condition of hyperthyroidism. Although his results are on the plus side, practically all are within accepted normal limits, especially if one considers the difficulty of attaining a so-called basal condition in young children. Our calculations from his data give as percentage variations from normal + 10, + 10, + 14, + 10, + 8 and + 17 per cent, respectively.

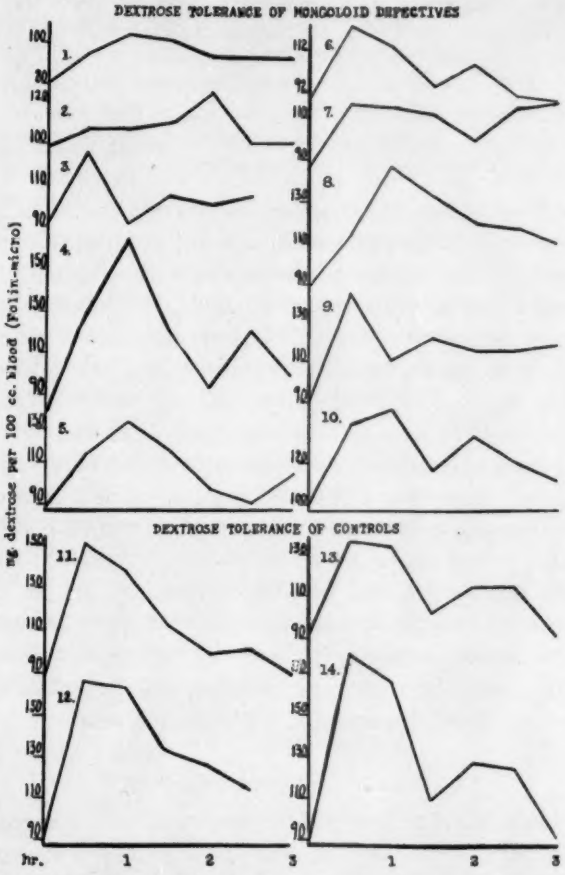
TABLE 8.—*Blood Sugar Values of Persons with Mongolism During Fasting*

Sex	Age Yr.	Age Mo.	Blood Sugar, Mg./100 Cc.	Sex	Age Yr.	Age Mo.	Blood Sugar, Mg./100 Cc.
F	5	3	92	F	13	6	95
M	5	4	69	F	14	2	83
M	6	5	93	F	14	8	94
M	6	10	75	F	14	11	109
M	6	11	85	F	15	0	97
M	7	2	111	F	15	0	84
M	7	5	96		15	1	94
M	7	6	85	F	15	1	104
M	7	7	88		15	2	96
F	7	8	78	M	15	6	83
	7	9	82		15	7	82
M	7	10	94	M	17	4	101
M	7	11	105	F	17	6	94
M	8	5	108	M	18	5	86
M	8	6	77	F	18	5	94
M	9	3	88	F	18	5	91
F	9	8	91		18	6	77
M	10	1	79	F	19	4	87
F	10	5	94	M	19	5	97
F	10	5	92	F	21	9	101
M	10	7	93	F	21	9	102
M	10	10	93	F	22	5	108
M	11	9	80		22	6	99
F	11	9	99	F	23	0	90
M	12	0	99	F	23	5	108
F	12	7	93	M	23	11	100
M	12	9	78	F	24	8	95
	12	10	84	F	28	5	95
M	13	4	77	F	29	0	113

When based on the Mayo Foundation standards, our results indicate a lower metabolism associated with a normal cholesterol level. Reports in the literature [33b] presumably based on these or similar standards have led to the conclusion that a low metabolic rate with a normal cholesterol level is associated with a "hypocondition" not due to the thyroid. We may assume, therefore, that our data support the view that mongolism is such a "hypocondition," perhaps a type of hypopituitarism. When the Talbot height standards are used, normal values are obtained. In any case, these basal metabolic rates with the normal cholesterol values rule out hyperthyroidism and hypothyroidism; they are not inconsistent with pituitary dysfunction.

DETERMINATION OF BLOOD SUGAR

Method.—Determinations of blood sugar were made on finger blood according to the micromethod of Folin.[39] One cubic centimeter of a 0.2 per cent solution of ferricyanide was used instead of 1 cc. or 2 cc. of a 0.4 per cent solution. This lessened the effect of a slight ferrous impurity, decreased the yellow color in the lower samples and allowed accurate results up to a maximum of about 200 mg. per hundred cubic centimeters. Analyses which gave above 150 mg.



Comparison of dextrose tolerance curves of 10 mongoloid subjects and of 4 nonmongoloid feebleminded controls.

Graph	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Sex	F	M	M	M	F	F	M	F	F	F	F	M	M	F
Age, yr. and mo.	7^{4}	7^{9}	8^{2}	8^{6}	11^{2}	11^{5}	11^{6}	18^{6}	26^{2}	30	6^{9}	7^{2}	9^{10}	15^{6}

were repeated with smaller aliquots of filtrate. Each set of samples was controlled by a blank and a 50 mg. or 75 mg. standard besides two 100 mg. standards.

39. Folin, O.: New England J. Med. **206**:727, 1932.

For the dextrose tolerance test the patients were kept in bed without breakfast. One and seventy-five hundredths grams of dextrose per kilogram of body weight was the dose used. It was given in sufficient water for easy solution, the concentration probably being less than 50 per cent, and was followed at once by a couple of swallows of water. When possible, specimens of urine were collected before the administration of the dextrose and each hour thereafter during the test. Triplicate samples of finger blood were taken before the dextrose was given and each half hour thereafter for three hours. In order that "bounces" in the tolerance curves might not be attributed to faulty circulation, the check samples were frequently taken from the fingers of different hands. The analyses were first made on duplicate samples, these being repeated and the triplicate values being determined if results did not check sufficiently closely the first time. Occasionally there were differences of 5 per cent, but most duplicate analyses agreed within 3 per cent or less. These careful checks made it certain that the interesting dips in the curves are real and not technical.

Results.—The fasting blood sugar values of 51 persons with mongolism aged from 5 to 29 years were normal, ranging from 69 to 113 mg. per hundred cubic centimeters of capillary blood (table 8). However, dextrose tolerance tests on 10 mongoloid children (chart) indicated a delayed glycemic response. There seems to be a tendency for a late peak, for a low curve, for a slow return to the fasting level or for a combination of these. The single curve (9) with a fairly high peak at the half hour interval was still 28 mg. above the fasting level after three hours. In none of these cases was sugar found in the urine. In a case (4) in which there was a high late peak no urine was voided. For comparison, tolerance curves of 4 nonmongoloid feebleminded controls are presented.

These results are consistent with hypofunction of the pituitary, since it is said [35] that in hypopituitarism there is frequently a diminution of the alimentary glycemic response but no uniform effect on the carbohydrate metabolism. The tolerance curves resemble closely those reported in the literature [40] for adolescents and adults having pituitary disturbance.

SUMMARY AND CONCLUSIONS

A study of the blood groups of 125 Americans with mongolism shows that the distribution corresponds to that of the general American population. This adds to the evidence that there is no relation between mongolism and the Mongolian race, thereby opposing the hypothesis of Down and Crookshank that mongoloid defect is due to racial regression.

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40. John, H. J.: Endocrinology **9**:397, 1925. Hamman, L., and Hirschman, I. I.: Studies on Blood Sugar: I. Alimentary Hyperglycemia and Glycosuria as a Test of Sugar Tolerance, Arch. Int. Med. **20**:761 (Nov.) 1917. Janney, N. W., and Isaacson, V. I.: The Blood Sugar in Thyroid and Other Endocrine Diseases: The Significance of Hypoglycemia and the Delayed Blood Sugar Curve, ibid. **22**:160 (Aug.) 1918. Gardner-Hill, H.; Jones, I., and Smith, J. F.: Quart. J. Med. **18**:309, 1925.

A brief survey is given of our studies on the pathology of the thyroid and pituitary glands and of the literature on the physiology of mongolism.

The color development in the Bloor method for the determination of cholesterol is discussed, and a modified technic is presented in detail.

The fasting serum cholesterol values for 50 persons with mongolism aged from 2 to 29 years are within normal range. They contrast sharply with the high values here recorded for 5 cretins. These results indicate that mongolism is not essentially a hypothyroid or a hyperthyroid condition. The proportion of esterified cholesterol in whole blood and in serum in 5 cases is also normal.

The basal metabolic rates of 25 persons with mongolism aged from 6 to 29 years are reported. They were calculated according to the Mayo Foundation standards and according to the Talbot height and weight standards. All are minus when reckoned by the Mayo standards, but all are well within normal limits when the Talbot height standards are used. The results are in contrast with the consistently low values found in 3 cases of cretinism. Here again the evidence indicates that in this age group the thyroid does not play a fundamental role.

The fasting blood sugar values of 51 persons with mongolism were found to be normal.

Dextrose tolerance tests were made on 10 mongoloid patients. The curves indicate delayed glycemic response suggestive of pituitary hypofunction.

Biochemical evidence indicates that thyroid failure is not of primary importance in mongolism, at least after the second year. Normal cholesterol levels, low or normal metabolic rates and normal fasting blood sugar values but low or delayed dextrose tolerance curves tend to confirm the assumption previously made that hypofunction of the pituitary is an important factor in mongolism.

Case Reports

SEVERE HEMORRHAGE SECONDARY TO RETROPHARYNGEAL ABSCESS

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A rare but often fatal complication of retropharyngeal abscess is hemorrhage due to erosion of the carotid artery or of one of its large branches. In the course of my training in otolaryngology a child aged 6 years was brought into the hospital on account of bleeding from the nose. She had been in poor health for two months. The lymph nodes on both sides of the neck had been enlarged for two weeks. Bleeding from the nose had been noted for the preceding twenty-four hours. A mass having a granular appearance was present in the nasopharynx. The leukocyte count was very high, and there were many immature cells. Consultants differed as to whether the condition was retropharyngeal abscess, malignant tumor or blood dyscrasia. Shortly after her admission to the hospital the child died of sudden profuse hemorrhage. At necropsy a small abscess high on the posterior pharyngeal wall was observed. It had eroded the carotid artery.

A few years later I received from another physician information regarding a somewhat similar case. This physician was called to see a child aged 2 years who recently had had scarlet fever. The lymph nodes of the neck were enlarged, and a large, fluctuant retropharyngeal mass was producing severe respiratory difficulty. There was nothing to suggest to the physician that the retropharyngeal mass might be anything other than an abscess, and on account of the severe dyspnea immediate drainage seemed imperative. Drainage was done in the approved manner, but the incision was followed instantly by uncontrollable hemorrhage, "as if the mass had been an aneurysm." Permission for necropsy was refused. Apparently, however, the child had had a retropharyngeal abscess which had eroded a large vessel, probably the carotid artery.

In the following case, which was similar, treatment was successful.

REPORT OF A CASE

A girl aged 9 years was brought to the clinic by her family physician on March 11, 1938. When the physician had seen the child in his office, on March 6, there were slightly enlarged lymph nodes in the right side of the neck and slight swelling on the right posterior pharyngeal wall, but the girl made no complaint. The physician had seen the patient at her home on March 9 and had found that the swelling both in the neck and in the posterior pharyngeal wall had increased

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slightly. There had been a little bleeding from the mouth for several hours. On March 10 there was a little further increase in swelling, and slight bleeding had continued intermittently. On March 11 the physician found that the swelling of the posterior pharyngeal wall had considerably more than doubled overnight. The bleeding had continued intermittently and at times had been profuse.

At the time of the patient's admission to the hospital the lymph nodes in the right upper cervical region were enlarged. A doughy mass involved the posterior pharyngeal wall on the right and extended down to the level of the base of the tonsillar fossa. There was no bleeding at the time of examination, but a considerable amount of old blood was visible in the mouth, pharynx and nose. The child appeared very ill. It was learned that mild scarlet fever had been prevalent in the community from which the child came and that she had had an illness one month previously for which a physician had not been called but which might have been scarlet fever. This suspicion was strengthened when urinalysis revealed evidence of nephritis.

The right common carotid artery was ligated. Because the child's condition was poor and the pharyngeal mass was draining through a small, spontaneously opened sinus and was not interfering with respiration, the mass was not incised. Appropriate supportive measures included transfusion of blood from the father. No further bleeding occurred. The child was better the following morning and on the second morning after operation sat up in bed to read the "funny papers." The pharyngeal mass diminished steadily. It was never incised. The patient was returned to the care of her private physician on the thirteenth day. At this time there was little evidence of the pharyngeal mass, and enlargement of the lymph nodes in the right upper cervical region was steadily receding.

COMMENT

An article by Richards [1] on retropharyngeal abscess deserves careful reading, particularly by general practitioners, pediatricians and otolaryngologists. He stated that a survey of a group of reports "at once brings to light the fact that the mortality is far greater than one would at first anticipate" (at least 7.4 per cent). In citing unusual causes of death, he stated that children with retropharyngeal abscess have been known to die suddenly from no other procedure than examination. Lifschutz [2] collected reports of 23 cases of hemorrhage secondary to retropharyngeal abscess, with 19 deaths. Of the 4 patients who recovered, 3 were treated by ligation and 1 recovered without ligation. Skoog [3] reported a case in which a peculiar "dark red-violet" discoloration of the mucous membrane over a fluctuant swelling of the posterior pharyngeal wall aroused his suspicion that the mass might contain

1. Richards, L.: Retropharyngeal Abscess, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1936, pp. 162-184.

2. Lifschutz, J.: Fatal Hemorrhage Resulting from the Extension of a Retropharyngeal Abscess to the Deep Vessels of the Neck, Arch. Otolaryng. **14**:149-157 (Aug.) 1931.

3. Skoog, T.: Septische Gefässarrosion im Carotisgebiete; einen Retropharyngealabszess vortäuschend, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:359-363 (July 22) 1933.

blood. Aspiration confirmed his suspicion. He placed a ligature around the carotid artery without tying it, then incised the mass and evacuated blood and old clots. No bleeding followed this procedure, and the loose ligature around the carotid artery was withdrawn after a few days.

Bleeding from or in association with a retropharyngeal abscess is rare, but when present warns of impending disaster. The carotid artery on the side of occurrence of the abscess should be ligated at once. Most patients who require this procedure are young children, and the risk of circulatory cerebral complications secondary to the ligation is slight in comparison with the danger of fatal hemorrhage without it. Rapid enlargement of a retropharyngeal mass in a few hours, as in the case reported, I believe to be significant. It may mean erosion of a large vessel even though no blood appears on the surface. If such a condition is suspected, aspiration before incision is wise.

CONGENITAL BILATERAL RADIO-ULNAR SYNOSTOSIS

ALMON R. CROSS, M.D.
DURHAM, N. C.

Radio-ulnar synostosis is a rare congenital bony union of the proximal ends of the radius and ulna, the lower ends being free. Supination is greatly impaired, the hand being held in constant pronation, but the condition is painless.

REPORT OF A CASE

H. C., a 7 year old boy, was admitted to the Duke Hospital on Jan. 2, 1939. The family history and past history were irrelevant. Since the age of 2 years the child's movements had been awkward when he took any article in his hands, and he could not turn the palms upward. There was no interference with flexion at the elbows. Except that both forearms were fixed in almost complete pronation and could not be supinated, the results of the physical examination were essentially negative. The child was able to write fairly well, but he had considerable difficulty trying to supinate the forearms to receive articles with the palms upward (fig. 1). Roentgen study of both elbows showed complete fusion of the radius and ulna of each arm at their proximal ends (fig. 2). The other long bones and the thoracic and abdominal viscera were examined fluoroscopically, but no other anomaly could be found. The Wassermann and Kahn reactions were negative.

COMMENT

Sandifort first described the condition in 1793 and reported 3 cases. To date 200 cases have been reported. The condition is congenital and sometimes familial. It may involve one or both forearms, producing a typical clinical picture consisting of fixation of the forearms in pronation. Supination is impossible except with rotation of the humeri. Attempts to correct these deformities surgically have not proved successful.

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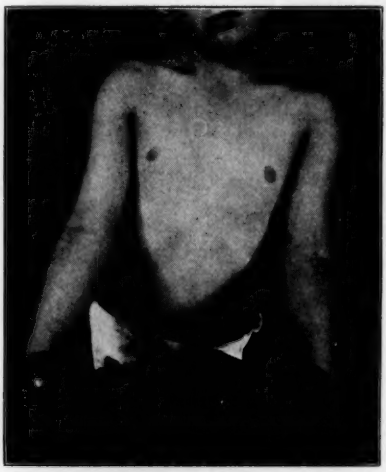

Fig. 1.—Photograph of the patient, showing inability to supinate the forearms.

Fig. 2.—Roentgenograms showing complete fusion of the radius and ulna of each forearm.

PLEURAL MANIFESTATIONS OF PERFORATION OF THE ESOPHAGUS

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BROOKLYN

Perforation of the esophagus in children, though infrequent, usually follows the accidental swallowing of almost any sort of object. Studies of foreign bodies in the air and the food passages [1] showed that most of the objects were not food particles. Although spontaneous rupture of the esophagus may occur, it is extremely rare.[2]

The diagnosis of perforation of the esophagus is often only inferred, even after careful endoscopic examination. Prudence demands that any mucosal injury be viewed with suspicion and that the patient be followed by frequent roentgen studies. Guthrie and Holland [3] formulated some roentgen criteria for the presence of a foreign body or abrasion of the esophageal mucosa, namely, (1) "hold-up" of swallowed opaque material, usually associated with some regurgitation, and (2) division of the medium into two streams which reunite lower down. McGibbon and Mather [4] noted the "hold-up" of a small piece of cotton wool impregnated with barium sulfate paste. If perforation has occurred, air may be present in the paraesophageal spaces,[5] and the esophagus may be displaced. Ingested iodized poppyseed oil may pass from the

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From the Department of Pediatrics, Israel-Zion Hospital, service of Dr. J. M. Wallfield.

1. Witkowsky, B.: Statistique d'une série de 250 corps étrangers de l'œsophage, Bronchoscop., œsophagoscop. et gastroscop., January 1937, p. 78. Vinson, P. P.: Foreign Bodies in the Food and Air Passages, M. Clin. North America **16**:1471, 1933.

2. (*a*) Gott, R., Jr.: Spontaneous Rupture of Esophagus, with Report of Four Cases, Am. J. M. Sc. **186**:400, 1933. (*b*) Fitz, 1877, cited by Gott.[2a] (*c*) March Goday, J.: Observacions sobre un cas de perforació esofàgica espontània, But. Soç. catalana de pediat. **5**:218, 1932. (*d*) Williams, T. H., and Boyd, W.: Spontaneous Rupture of the Esophagus, Surg., Gynec. & Obst. **42**:57, 1926.

3. Guthrie, T., and Holland, C. T.: Some Observations on Meat and Fish Bones Impacted in the Esophagus, J. Laryng. & Otol. **38**:229, 1923.

4. McGibbon, J. E. G., and Mather, J. H.: Perforation of the Esophagus by Swallowed Foreign Bodies, Lancet **2**:593, 1935.

5. Iglauer, S., and Ransohoff, J. L.: Perforation of Esophagus by Foreign Body, Laryngoscope **34**:821, 1924.

esophagus into the tracheobronchial tree or into the pleural cavity,[6] as was demonstrated in the case to be reported.

The prognosis of any esophageal perforation must be considered as grave, and Jackson is quoted [7] as saying that the esophagus is surgically the most intolerant organ in the body and that a foreign body lodged there may quickly prove fatal, death resulting from hemorrhage, from asphyxia or from perforation and septic mediastinitis.

According to Williams and Boyd,[2d] treatment consists of putting the esophagus completely at rest by gastrostomy and of early thoracotomy with the open method whenever pleural manifestations are present.

REPORT OF A CASE

History.—F. W., a girl aged 3 years, was admitted to the children's ward of Israel-Zion Hospital on Jan. 15, 1935, with a history of cough, fever, rapid breathing, pain in the chest and general malaise of three weeks' standing. The family history was irrelevant. The child was born at full term with forceps delivery. Convulsions occurred at the age of 10 months, with residual weakness of the right side of the face. Frequent colds were also noted.

The present illness began on Dec. 25, 1934, with an infection of the upper respiratory tract and a rash; the diagnosis was possible measles. Four days later vomiting, high fever, cough and abdominal pain were present; at first acute appendicitis was suspected, but the diagnosis was later changed to pneumonia. The temperature remained septic, ranging from 102 to 104 F., and on Jan. 8, 1935, fourteen days after the onset, the child began to expectorate a moderate amount of purulent, malodorous sputum. A ruptured pulmonary abscess was now suspected. The chest, as reported by the family physician, had showed flatness over the right side up to this time, but this seemed to diminish during the following week, and the numerous moist rales apparently pointed to resolution. On January 15 the temperature rose to 104.5 F., and hospitalization was ordered.

Physical Examination.—On admission the child was acutely ill; she lay on the right side, and there was some cyanosis of the lips and finger nails. The temperature was 103 F., the pulse rate 160 and the respiratory rate 64. The heart was displaced markedly to the left; the sounds were of fair quality, and there was no evidence of pericarditis. Examination of the lungs revealed dulness merging into the flatness over the right pulmonary field and some dulness at the base of the left lung. Breath sounds were distant and bronchial over the right side of the chest, and a questionable succussion splash was elicited on the right. The abdomen was distended, with some resistance and tenderness on pressure over the hepatic region. The liver was palpable 3 fingerbreadths below the costal margin. The neurologic status was normal. The diagnosis was pyopneumothorax on the right, with a possible additional underlying abscess of the lung.

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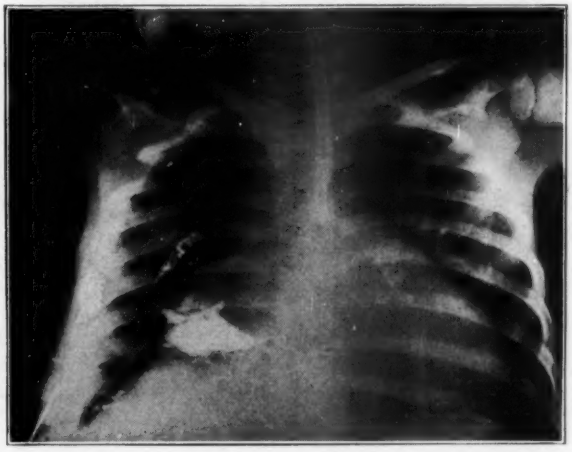
6. Mikula, cited by Mörl, F.: Oesophagusperforationen in die Pleurahöhle, Beitr. z. klin. Chir. **158**:487, 1933. Iglauer, S., in discussion on Orton, H. B.: Mediastinitis Following Esophageal Foreign Body: Report of Cases, Arch. Otolaryng. **12**:845 (Dec.) 1930.

7. Jackson, C., cited by Orton, H. B.: Mediastinitis Following Esophageal Foreign Body: Report of Cases, Arch. Otolaryng. **12**:635 (Nov.) 1930.

Treatment and Course.—A roentgenogram of the chest taken before aspiration showed hydropneumothorax involving the greater portion of the right side. Thoracentesis was done, and 40 cc. of milky, yellowish brown purulent fluid was obtained. There was no change in the patient's condition during the next four days; the temperature ranged from 101 to 102 F. Fluoroscopic examination at this time showed the right pleural cavity to be half full of fluid, with air above and collapse of the right lung. No cavity was seen in the lung.

At this time Dr. Rosenthal, director of laboratories, reported the presence of yeast and Bacillus acidophilus in fluid obtained from the chest by aspiration; he suggested the possibility of a communication between the gastrointestinal tract and the pleural cavity.

With the patient under general anesthesia, open thoracotomy was performed the next day by Dr. B. Jaffe, and 2½ inches (6.3 cm.) of the eighth rib was removed in the postaxillary line on the right side. There was profuse drainage of fibrinopurulent material. Two hundred and fifty cubic centimeters of whole



Esophagopleural fistula with iodized poppyseed oil entering pleural cavity.

blood was given by transfusion two days later because of a drop in the hemoglobin content from 70 per cent (on admission) to 55 per cent and a drop in the red cell count from 3,900,000 to 2,200,000. It was now noticed that some methylrosaniline (gentian violet) which had been applied to an oral lesion colored the dressing of the empyema blue. The following day some particles resembling curdled milk and a few shreds of undigested string beans were found on the dressing, and a diagnosis of perforation of the esophagus with pleural fistula and sitothorax (food in the pleural cavity) was self evident. A Levine tube was passed into the stomach without difficulty, and high caloric liquid feedings were given through it. Drainage from the chest decreased immediately after insertion of the tube, and instead of the puriform material, a thick mucoid discharge was present.

Fluoroscopic study now showed little fluid in the right pleural cavity and only partial collapse of the right lung. Under the fluoroscopic screen iodized poppyseed oil, given by mouth, could be seen descending alongside the Levine tube to the lower part of the esophagus just above the diaphragm, where it divided into two streams, one going into the stomach and the other turning off at a right angle

to enter the right pleural cavity. A roentgenogram made at this time showed an esophagopleural fistula at the level of the eighth thoracic vertebra, some of the oil being mixed with fluid in the right pleural cavity. The right lung was still collapsed. No diverticulum or foreign body was noted (figure).

Gastrostomy was done with the area under local anesthesia, with the hope that the fistula would heal spontaneously. A 5 per cent solution of dextrose was given by continuous intravenous drip. The temperature rose to 104.8 F. the next day, with no increase in pulse and respiratory rate. Slight cyanosis of the lips and finger nails was present, and oxygen therapy was begun. The abdomen was soft and only slightly distended. The general condition of the patient was poor, however, and she died on the following night.

At this time an addenda to the history was obtained from the mother: On December 25, the day of onset of the present illness, the child had choked and coughed during a meal of chicken and was supposed to have swallowed a small bone.

Laboratory Observations.—On the patient's admission the hemoglobin content was 70 per cent; red cells numbered 3,900,000 and white cells 26,200; there were 87 per cent polymorphonuclears and 13 per cent lymphocytes. The Schilling differential count showed 6 stab cells, 4 young cells and 3 myelocytes. The hemoglobin content and the red cell count dropped to low levels but rose after the transfusion. The urine contained a faint trace of albumin and an occasional white cell. Culture of the thoracic fluid yielded yeast and B. acidophilus.

The final clinical diagnosis was esophagopleural fistula following perforation of the esophagus, sitothorax (food in the pleural cavity), secondary pyopneumothorax, open thoracotomy (right) and gastrostomy.

COMMENT

The diagnosis of a perforated esophagus was made in this case only after food particles were discharged from the thoracotomy opening and confirmatory studies with iodized poppyseed oil were made. The culture of B. acidophilus from material obtained by thoracentesis is, in retrospect, an indication of acid contents in the pleural cavity and should serve as a sign of a possible esophagopleural communication.

SUMMARY

A case of perforation of the esophagus is reported; this was associated with an esophagopleural fistula, food particles in the pleural cavity (sitothorax) and secondary pyopneumothorax but not with diffuse suppurative mediastinitis.

The thoracic tap showed B. acidophilus, which indicated a communication with the digestive tract.

Ingestion of iodized poppyseed oil demonstrated the branching of the stream from the esophagus into the pleural cavity.

In spite of both thoracotomy and gastrostomy, the patient died.

PNEUMONIA COMPLICATED BY AN ACUTE HEMORRHAGE AND ULCERATIVE GASTRO-ENTERITIS IN A CHILD

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AND
ALLAN BLOXSOM, M.D.
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The rarity of a large hemorrhage with ulcerative gastroenteritis accompanying pneumonia in a child warrants the following case report.

REPORT OF A CASE

T. H., a white boy aged 12 years, was admitted to the pediatric service of the Memorial Hospital on Oct. 14, 1938, because of vomiting and passing of blood.

The mother stated that the onset of the infection occurred five days previous to the time of admission, when the patient had a cold with some general discomfort. On the next day he had a severe headache and a temperature of 102.2 F. He was constipated, had severe epigastric pain and began to vomit all his food. On the next day the temperature rose to 103 F. It remained at that level until his admission to the hospital.

The family physician gave the patient mild mercurous chloride on the third day of the illness, and the boy began to pass watery stools. There was no blood in the stools at this time.

On the morning of admission to the hospital the patient suddenly coughed and brought up a mouthful of bright red blood. The following morning he passed several dark and tarry stools and vomited twice, each time bringing up material resembling coffee grounds. Shortly before admission in the afternoon the boy again coughed bright red blood and vomited dark material twice.

The patient's mother, one aunt and one uncle had tuberculosis.

The boy always had been underweight and undernourished. As an infant he was breast fed. He walked at 19 months of age, was slow in learning to talk and at the time of his admission to the hospital just beginning to read. The parents stated that he had presented a "feeding problem" all his life, being extremely sensitive to a number of green vegetables. Almost every summer up to 10 years of age the child had had diarrhea, accompanied with bloody mucus and considerable tenesmus. The attacks usually lasted several weeks. At 7 years of age he had had bilateral pneumonia accompanied with dysentery.

On physical examination the boy was seen to be acutely ill. The skin was ashen blue; he was extremely slender, and his lower jaw was prominent. His temperature on admission was 103.8 F.; the pulse rate was 130 and the respiratory rate 22. Besides the marked ashen blue appearance of the skin there was severe herpes labialis. The tongue was thickened and covered with a thick white coat. The breath sounds were suppressed over the lower lobe of the left lung, with a

From the Pediatric Staff of the Memorial Hospital, Houston, Texas.

few fine rales in this area. The abdomen was distended. There was no rigidity or tenderness. The extremities were poorly developed, and the muscle tone was weak.

A roentgenogram of the chest revealed a central consolidation of the lower lobe of the left lung. Examination of the blood showed a leukocyte count of 21,000 per cubic millimeter, with polymorphonuclears 90 per cent and lymphocytes 10 per cent. The red blood cell count was 2,000,000 per cubic millimeter, and the hemoglobin content was 36 per cent. Urinalysis gave essentially normal results. The Widal test, the Wassermann test and the blood culture gave negative results. Examination of a smear from the blood showed no malaria. Examination of the stool showed many red blood cells and a large number of gram-positive diplococci. Agglutination of the bacteria obtained from the stomach and from the sputum was negative for pneumococci of types I to IX.

A guinea pig was inoculated with the sputum after the Mantoux test with 10 mg. of old tuberculin had given a negative result. The guinea pig showed no signs of tuberculosis at autopsy. Fluoroscopic and roentgenographic examinations of the gastrointestinal tract were made, and no pathologic condition could be demonstrated.

The coagulation time was three minutes; the bleeding time, eight minutes; the clot retraction time, one hour and four minutes, and the platelet count, 200,000.

Treatment consisted in transfusions, administration of fluids and absolute rest.

The patient apparently had no further bleeding after eight hours, about four hours after the first transfusion. The temperature ranged from 99 to 105 F. for two days, falling to normal by lysis on the ninth day after admission. The respiratory rate varied from 20 to 30 and the pulse rate from 100 to 130. The child was discharged on the thirteenth day after admission. Four days before his discharge the red blood cell count was 2,760,000 per cubic millimeter, the hemoglobin content 65 per cent and the leukocyte count 21,000 per cubic millimeter, with the following differential count: lymphocytes, 22 per cent; polymorphonuclears, 76 per cent, and eosinophils, 2 per cent.

COMMENT

Johnson [1] reported in 1929 the case of a 12 year old boy who had symptoms of influenza accompanied by the passing of almost a pint of blood. There was no hemoptysis or hematemesis.

Sanford, Hughes and Weems [2] reported 2 cases in which pneumonia in adults was complicated by acute pneumococcic hemorrhagic ulcerative gastroenteritis. They reviewed the literature, which contains 8 cases in addition to the case reported by Johnson. In both of the cases reported by Sanford, Hughes and Weems type IV pneumococci were present in the sputum.

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1. Johnson, W. M.: Intestinal Hemorrhage as a Complication of Pneumonia, Arch. Pediat. **46**:193 (March) 1929.

2. Sanford, C. H.; Hughes, J. D., and Weems, J.: Pneumonia Complicated by Acute Pneumococcic Hemorrhagic Ulcerative Gastroenteritis (Dieulafoy's Erosion), Arch. Int. Med. **62**:597 (Oct.) 1938.

The case reported here is unusual in that the child had a hemorrhage from the lungs in addition to the hemorrhages from the gastrointestinal tract.

Pneumococci recovered from the sputum and from the stomach were not agglutinated by serums I to IX.

SUMMARY

A case of pneumonia complicated by hemoptysis, hematemesis and melena in a child is reported. The case is, as far as we can determine, the first of its kind to be recorded in the literature.

CONGENITAL LEUKEMIA

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AND
DOROTHY H. ANDERSEN, M.D.
NEW YORK

In 1922 Koch [1] described leukemia in a stillborn premature infant. Since that time there have appeared sporadic case reports of congenital leukemia. Büngeler [2] pointed out some of the difficulties of diagnosing leukemia in the newborn; he emphasized the failure of earlier writers to differentiate between erythroblastosis foetalis and leukemia and stated that certain cases of von Jaksch anemia may also have been a source of confusion. Kornmann [3] collected 5 cases previously described and added a sixth. The present report summarizes the literature and presents an additional case of congenital leukemia.

REPORT OF A CASE

R. B. A., a boy, was admitted to the Babies Hospital when 30 days old with a history of purpura and fever of seven days' duration. The parents were well. There was no family history of a tendency to bleed. The patient was the result of the first pregnancy. Birth was effected with forceps. The birth weight was 2.64 Kg. At birth the child appeared normal except for a forceps mark over the right temporal region. He remained well until the onset of purpura twenty-three days after birth. On his admission to the hospital the physical examination revealed a desperately ill child with a temperature of 103.8 F. and a pulse rate of 190. Massive ecchymoses covered the whole body. Numerous hemorrhagic bullae about 1 cm. in diameter were scattered over the arms and scalp; some of these had ruptured and become infected. Occasional small petechiae were found in the mucous membranes. The superficial lymph glands were enlarged everywhere, varying in diameter from several millimeters to 1.5 cm. The heart seemed normal except for marked tachycardia. There were equivocal signs of consolidation in the upper pulmonary field on the right. The abdomen was practically filled by the liver and spleen, the former reaching almost to the symphysis pubis

From the Babies Hospital and the Departments of Diseases of Children and Pathology, College of Physicians and Surgeons, Columbia University.

1. Koch: Zur Frage der kongenitalen Leukämie, Zentralbl. f. allg. Path. u. path. Anat. **33**:7, 1922.

2. Büngeler, W.: Angeborene Leukämie, Frankfurt. Ztschr. f. Path. **41**: 257, 1931.

3. Kornmann, V.: Beitrag zur frühkindlichen und angeborenen myeloischen Leukämie, Ztschr. f. Kinderh. **56**:440, 1934.

and the latter extending to the iliac crest. Examination of the blood showed 6.5 Gm. of hemoglobin per hundred cubic centimeters, 2,000,000 red blood cells and 223,500 white blood cells per cubic millimeter, 2 per cent polymorphonuclear cells, 22 per cent myeloblastic cells, 68 per cent myelocytic cells and 2 per cent lymphocytic cells. Platelets numbered 110,000 per cubic millimeter. The bleeding time was longer than thirty minutes. The clotting time was two minutes. The peroxidase stain demonstrated granules in 7.5 per cent of the cells. The Kline test gave a negative reaction. The urine was clear. The blood culture remained sterile. Stools were black, and the guaiac test gave a 2 plus reaction. Roentgenograms of the long bones and skull showed no changes that could be interpreted as due to leukemia. There was slight irregularity of the metacarpal bones. Areas of diminished density were found in the distal portions of the long bones by roentgen examination. The child became progressively more dyspneic, more purpuric and weaker; he died two days after admission. He appeared unusually toxic. No neurologic signs, cardiac failure or evidence of consolidation developed, so that failure of a definite organ could not be named as the cause of death.

Autopsy.—Gross Examination: The infant was well developed and nourished, weighing 4.6 Kg. and measuring 55.5 cm. in length. The striking feature of the body was the presence of widely disseminated lesions of the skin. These were of three types. Deep ulcers extending through the epidermis and covered with clean hard crusts were noted over the right frontal bone, the occiput and the left temporomandibular joint; these measured 1 to 3 cm. in diameter. Similar smaller ulcers were observed over the scalp, the face, the breast and the inner surface of the left arm. The second type of lesion was composed of a minute yellowish white dot surrounded by a halo of hemorrhage, the whole measuring 5 to 10 mm. in diameter. The third lesion was diffuse ecchymotic mottling. The two lesions last described were spread over the hand, the chest, the upper parts of the arms and the thighs. The forearms and the lower portions of the legs were relatively normal. The contours of the chest were normal. The abdomen was protuberant. The mucous membranes appeared normal. The subcutaneous fat was edematous and of normal amount. The umbilical vessels appeared normal. There was slight diastasis recti. The abdominal distention was due to the large liver, which extended 7.5 cm. below the costal margin. The spleen lay behind the left lobe of the liver. The pleural and pericardial cavities appeared normal. There was a diffuse hemorrhage in the neck, anterior to the trachea, extending from the hyoid bone to the sternum.

The heart weighed 28 Gm. There were countless petechial hemorrhages, which were nearly confluent over the right auricular appendage and were scattered in smaller numbers over the aorta and along the coronary arteries. The largest, measuring 3 mm., had a gray center and lay over the apex. There were several petechiae in the myocardium. The foramen ovale was patent by a narrow slit. The heart was otherwise normal.

The right lung weighed 57 Gm. and the left 49 Gm. Both lungs were crepitant. Many small hemorrhages were present, some of which had grayish centers. Each involved one to several lobes. The cut surface was edematous, and the bronchi contained foamy fluid.

The spleen weighed 48 Gm. and measured 8 by 5.5 by 3.7 cm. The margins were rounded, and the surface was smooth. The cut surface was pale grayish red, soft and homogeneous. The malpighian corpuscles were not visible.

The liver weighed 410 Gm. and measured 14.5 by 11.5 by 4.5 cm. The surface was smooth, and the margins were rounded. The color was pale, with slight mottling, and close inspection showed scattered, poorly demarcated gray areas less

than 1 mm. in diameter. The cut surface showed the portal areas to be pale gray and irregular in size. The gallbladder was normal.

The contours of the pancreas were normal, but on section many of the lobules were fused, and in these areas the tissue was firm and pale. The adrenals had a thin cortex and a hemorrhagic reticular zone.

The right kidney weighed 46 Gm. and measured 6.5 by 4 by 3 cm. The left kidney weighed 43 Gm. and measured 6.8 by 4 by 3 cm. The capsules stripped easily, leaving pale surfaces mottled with diffuse areas of congestion and also with many petechiae. On section the cortexes were pale and mottled; the pyramids were red. Both kidneys contained petechiae (fig. 1).

The bladder and prostate were normal. The testes were purplish red and on section were seen to contain small hemorrhages.

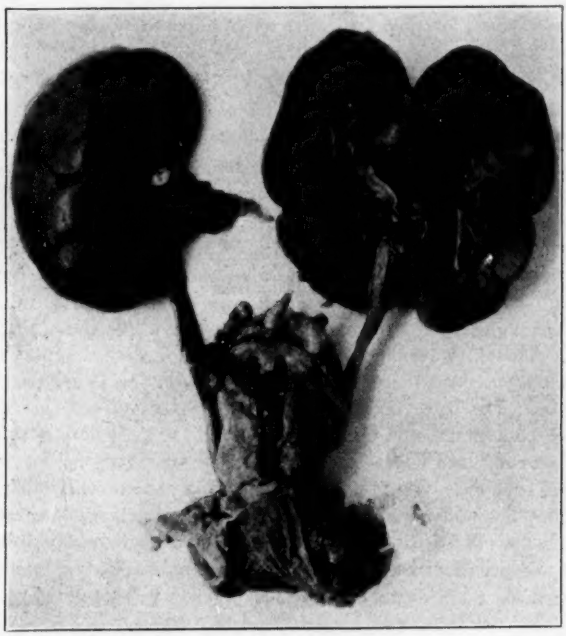

Fig 1.—The kidneys.

The esophagus was normal. The stomach bore petechiae over both the peritoneal and the mucosal surface. The small intestine appeared normal except for prominence of the lymph follicles and of Peyer's patches. The latter measured 1 or 2 mm. in thickness and contained petechiae. The peritoneal and mucosal surfaces of the appendix and colon contained mottled red areas corresponding to areas of hemorrhage in the lymph follicles.

The epiglottis was edematous. The tonsils were small and appeared normal. The larynx was normal. The tracheal mucosa was dull and congested. The thyroid was normal except for diffuse hemorrhage. The thymus was large, measuring 5.5 by 4.5 cm. It contained several gray nodules in which the lobular structure was obliterated. The largest of these was in the lower end of the right lobe and measured 1.5 cm. in diameter.

The cervical, axillary, inguinal, retroperitoneal, mesenteric and mediastinal lymph nodes were all moderately enlarged, measuring 5 to 10 mm. in diameter. The cut surface was homogeneous and gray, sometimes with a faint reddish brown tinge. Small hemorrhages were present. The largest nodes, those lying near the common duct, measured about 1.5 cm. in length.

Permission for examination of the brain was not received.

Microscopic Examination: The striking feature of the microscopic sections was an infiltration of large cells throughout the various organs (fig. 2). These cells had large, pale, finely granular nuclei, which were round or oval, and a scanty cytoplasm with a distinct cell membrane. Occasionally the nuclei were indistinct. A few myelocytes were noted among them. Oxidase stains showed that a few

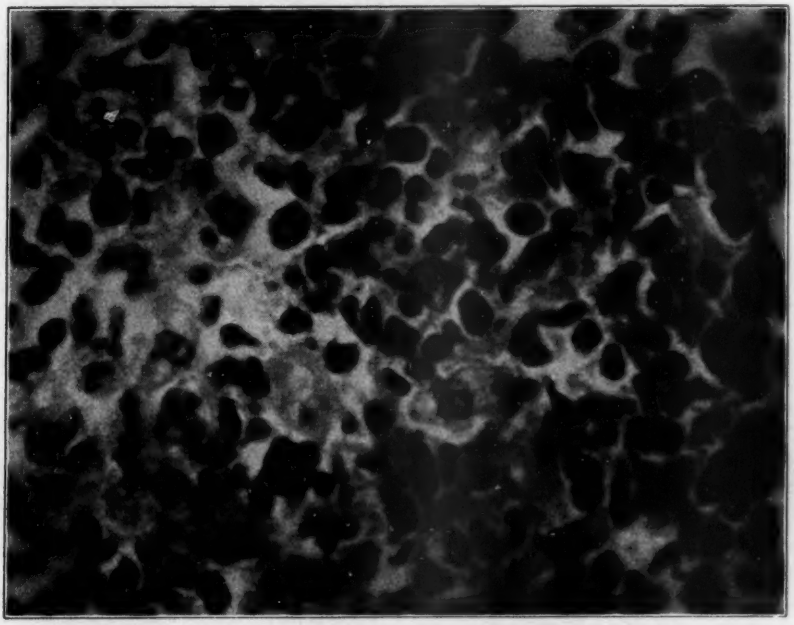

Fig. 2.—Leukemic infiltration in lymph nodes.

(approximately 5 per cent) of these large cells contained oxyphil granules, but the majority did not. Giemsa stains showed that the majority of the cells had a clear, faintly bluish cytoplasm, and in a small proportion of cells there were fine bluish granules. These cells were noted in the blood, and they infiltrated most of the organs.

The heart contained leukemic infiltrations in the epicardium and around small arteries in the myocardium.

In the lung infiltrations were present in the alveolar walls, around the bronchi and within the alveoli.

In the spleen the infiltrating cells occupied the pulp. The malpighian corpuscles were small.

The portal areas of the liver were greatly enlarged because of the infiltration of large cells. The sinuses were distended with similar cells, and the liver columns were narrowed.

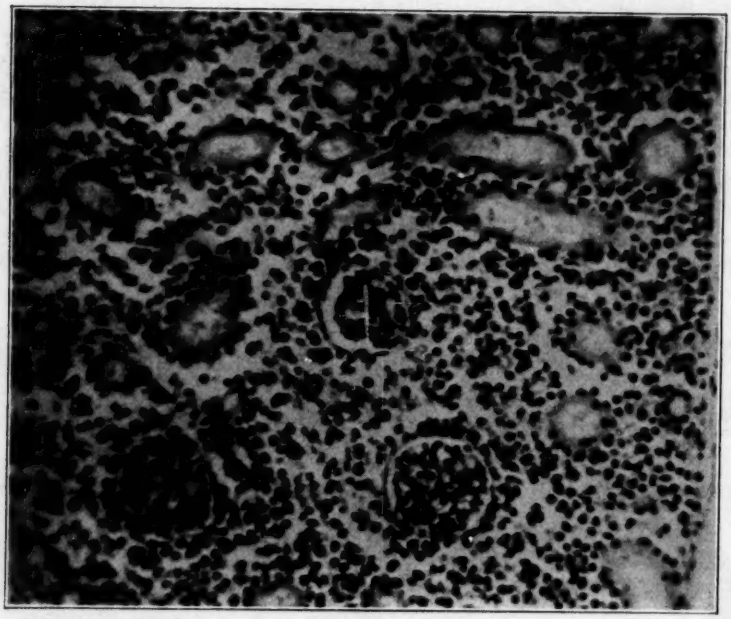

Fig. 3.—Kidney.

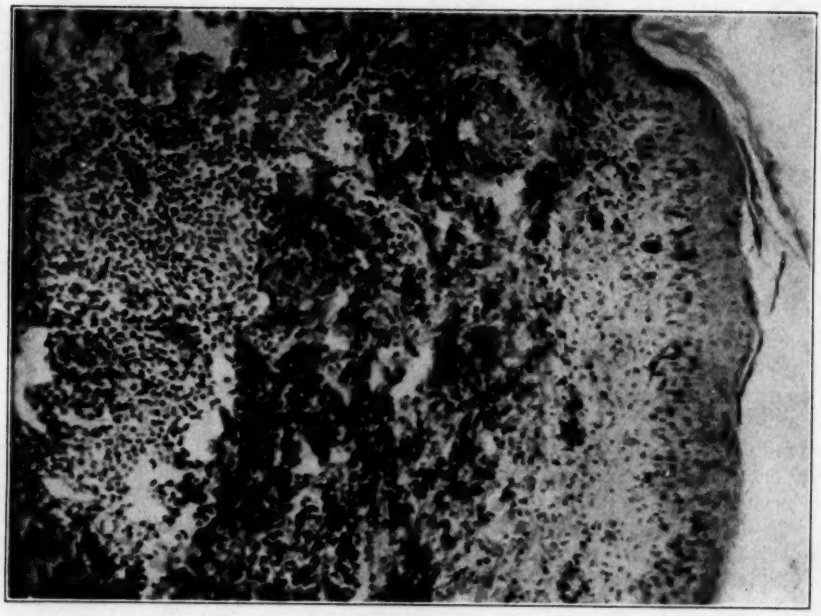

Fig. 4.—Skin.

The infiltrations involved the entire wall of the gallbladder, including mucosa, muscularis and peritoneum.

In the pancreas the abnormal cells occupied the interstitial tissue and replaced much of the parenchyma. The acini which remained appeared normal.

The inner portions of the adrenal cortex were occupied by the abnormal cells, and small groups were seen in the periadrenal connective tissue.

In the kidneys the glomeruli and tubules were separated from one another by wide areas of infiltrating cells, which lay in the interstitial tissue and occupied at least three quarters of the cortex. The pyramids were less affected, but the pelvic mucosa was densely infiltrated (fig. 3).

The testis was densely infiltrated and also contained small hemorrhages.

The mucosa of the stomach contained only a small number of abnormal cells, but they densely infiltrated the intestinal mucosa and almost entirely replaced the lymphoid tissue.

The tissue of the lymph nodes and thymus was largely replaced by the large cells.

The skin contained infiltrations around the glands and hair follicles of the dermis (fig. 4). Similar cells were observed around small vessels in the subcutaneous fat and in the underlying striate muscle.

REVIEW OF LITERATURE

After a critical survey of the reported cases of congenital leukemia in the neonatal period, it was felt that they should be divided into three groups. The first group includes cases in which the diagnosis of leukemia appears to be firmly established. The minimum interval between the first appearance of leukemic cells in the tissues and blood and the first appearance of clinical symptoms of leukemia is unknown. Therefore there is absolute proof of the onset of the disease in intrauterine life only in the 2 cases in which it was observed in stillborn infants. Group 1 includes also cases in which either the onset of the disease was sufficiently early to justify the belief that the leukemia was congenital or the degree of infiltration appeared too great to have taken place within the child's extrauterine life. The second group is made up of cases in which the diagnosis of leukemia seems justifiable but the age of onset was uncertain. In these cases there was usually a vague history of ailments in the first weeks of life, but the pathognomonic symptoms came so late that the disease may well have begun after birth. The last group consists of a few cases in which illness was present at birth but the diagnosis could not be definitely established.

In addition to the case reported here, the first group includes the cases of Smith,[4] Koch,[1] Stransky,[5] Büngeler,[2] White and Burns,[6]

4. Smith, L. W.: A Case of Acute Leukemia in an Infant, Am. J. Dis. Child. **21**:163 (Feb.) 1921.

5. Stransky, E.: Beiträge zur klinischen Hämatologie im Säuglingsalter: II. Zur Frage der kongenitalen myeloischen Leukämie, Monatschr. f. Kinderh. **29**: 654, 1925.

6. White, P. J., and Burns, E. L.: Fatal Acute Lymphoblastic Leukemia with Great Enlargement of the Kidneys in an Infant Three Weeks Old, Am. J. Dis. Child. **41**:866 (April) 1931.

Giblin,[7] Kornmann [8] and Cooke.[8] The ages of onset of symptoms varied from newborn to 33 days. The oldest patient lived to be three months old. The family histories were irrelevant. No instance of leukemia in the parents was found. The chief complaint in 2 cases was "failure to do well," in 4 was hemorrhage and in 1 was vomiting and diarrhea. Two infants were stillborn. One patient had icterus neonatorum, and another had mild edema. In all cases enlargement of both spleen and liver was found. In all the lymph nodes were seen to be enlarged either clinically or at autopsy except in 2 instances, in neither of which autopsy was performed. Six patients had evidences of a hemorrhagic tendency, 2 had infections, 1 superficial omphalitis and the other terminal pneumonia. The blood pictures consistently showed diminution of the red cell count and hemoglobin level. Erythroblasts were present in the blood of 2 infants; in 1 case the count rose to 40,000 per cubic millimeter, the leukocyte count varying between 36,000 and 420,000. In the cases in which the platelet counts were mentioned they were low. The clotting time was normal in the 2 cases in which it was noted. The bleeding time was elevated in 2 of the 3 cases in which it was ascertained. No evidence of syphilis was found in any of the cases. Six complete autopsies were performed. One case is included in which autopsy was not obtained but in which the clinical findings fulfilled all requirements for a diagnosis of leukemia. In Giblin's case only the spleen was examined.[7] Only the kidneys were described in the case of White and Burns.[6] In all of the 6 cases in which complete autopsy was performed the lymph nodes, liver, spleen and kidneys were infiltrated with leukemic cells. Except in the case reported by White and Burns, when the observers felt certain of the type of leukemia the diagnosis was acute myeloid leukemia. The difficulties of determining the exact character of acute leukemia are well known, and there may have been more instances of lymphoid leukemia in this group. We feel that from the findings on the blood in the case of White and Burns one cannot make an unqualified diagnosis of lymphatic leukemia.

In cases reported by Opitz,[9] Tancré [10] and Malmberg [11] the diagnosis was accepted by the authors. These cases differ from those of

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7. Giblin, J.: A Case of Myelogenous Leukemia Occurring in an Infant Five Weeks Old, Arch. Pediat. **50**: 662, 1933.

8. Cooke, J. V.: Personal communication to the authors. In Cooke's patient a small lump developed on the sternum at the age of 10 days. This mass persisted, and the infant had fever and anorexia. Pallor and hemorrhagic spots appeared, together with lymphadenopathy and enlargement of the liver and spleen. Typical leukemic infiltrations were noted at autopsy.

9. Opitz, J.: Ein Fall von myeloischer Leukämie beim Säugling, Med. Klin. **20**:232, 1924.

the previously mentioned group in that the onset was indefinite and the life span longer. In Opitz' case the parents thought the child was pale and had a large abdomen at birth. The onset of the illness may have been as late as the age of 2½ months. Malmberg obtained a history of pallor and slight edema at birth but did not make his diagnosis until the child was 5½ months old. In Tancré's case the abdomen was thought to be enlarged at 4 weeks, but definite symptoms did not develop until 8 weeks after birth. The pathologic picture of this group was similar to that of the first group except in Tancré's case, in which there was no enlargement of the lymph glands. Nothing was found that would suggest syphilis in the patients. Malmberg and Opitz thought that their cases were instances of myeloid leukemia. In Tancré's case the character of the leukemia could not be definitely established.

The third group is composed of cases which were reported with the diagnosis of leukemia but in which the evidence for this diagnosis appears inadequate. Several of the early cases are included. Similar cases have been summarized by von Gierke.[12] Pollmann's[13] patient had clinical signs of a cardiac lesion, mild icterus and enlargement of the liver, spleen and lymph nodes. The leukocytes numbered 300,000 per cubic millimeter. Infiltration in the kidneys was described. Tricuspid endocarditis was observed at autopsy. This may have been an instance of an unusually high leukocytosis of infection. Lommel[14] described a child born with edema and a large abdomen. The spleen and liver were large. There were 1,240,000 leukocytes per cubic millimeter. The kidneys and liver were infiltrated. The difficulty in retrospective diagnosis lies in the brevity of the hematologic report, the lack of enlarged lymph glands and the presence of edema at birth. In Chapman and Ewart's[15] case the familial history of icterus, the number of erythroblasts, the small liver and spleen and the edema make probable the diagnosis of erythroblastosis foetalis. The only confusing point was a leukocyte count of 142,000 per cubic millimeter and a differential blood count suggestive of leukemia. The patient of Didier

10. Tancré, E.: Beitrag zur akuten lymphatischen Leukämie im Säuglingsalter, Arch. f. Kinderh. **67**:7, 1918.

11. Malmberg, N.: Beitrag zur Kenntnis der myeloiden Leukämie bei Säuglingen, Acta pædiat. **4**:410, 1924.

12. von Gierke, E.: Ueber foetale Erythroleukoblastose, Virchows Arch. f. path. Anat. **275**:330, 1930.

13. Pollmann, L.: Ein Fall von Leukämie beim Neugeborenen, München. med. Wchnschr. **45**:44, 1898.

14. Lommel, F.: Zur Behandlung der Leukämie und Pseudoleukämie mit Roentgenstrahlen, München. med. Wchnschr. **52**:904, 1905.

15. Chapman, S. G., and Ewart, I. B.: Acute Congenital Leucocythaemia, New Zealand M. J. **31**:19, 1932.

and Chomé[16] had jaundice and hemorrhages. There were 61,300 leukocytes per cubic millimeter, 50 per cent of which were early myeloid forms. The kidneys, liver and spleen were reported as infiltrated, though the lymph nodes were normal. With the presence of jaundice, 6,000 nucleated erythrocytes per cubic millimeter, an unusually low red cell count (800,000 per cubic millimeter) and the lack of lymph gland enlargement, we are inclined to believe that the condition in this case may have fallen more properly into the erythroblastosis foetalis–icterus gravis group and that the diagnosis of leukemia was uncertain. A recently reported case of the former condition is that of Rhamy.[17] No clinical findings justified the diagnosis of leukemia, on which the pathologists did not agree. It is interesting that there were protagonists for both erythroblastosis and leukemia. In no instance did a leukemic mother give birth to a leukemic infant. This is interesting, since certain tumors (sarcomas) have been reported as crossing the placenta. In 1 case fairly definite congenital lymphatic leukemia was found.

COMMENT

That congenital leukemia occurs seems acceptable. It is equally well recognized that the diagnosis of leukemia in early life is not always simple. Leukemoid states are not unusual with many severe infections in infancy. Many similarities exist between erythroblastosis foetalis and congenital leukemia. Both produce increased numbers of leukocytes, including early forms, usually of the myeloid series. Young forms of erythrocytes may appear in both diseases. Both usually cause hepatomegaly and splenomegaly. Various hemorrhagic manifestations are common to both. The presence of edema and of severe neonatal jaundice, the lack of general lymphadenopathy and the familial history of similar symptoms characterize erythroblastosis and are lacking in a case of leukemia. Infiltration of early leukocytic forms into non-hemopoietic organs is frequently seen with leukemia and never with erythroblastosis.

The criteria for the diagnosis of congenital leukemia are divided into clinical and pathologic data. If the clinician is to consider the disease congenital, the symptoms should be present either at or within a few days after birth. If he is to call the disease leukemia there should be enlargement of the liver, spleen and lymph nodes. The blood picture should show high leukocytosis, with early forms predominant. There should be nothing in the history to indicate syphilis, erythro-

16. Didier and Chomé: Sur un cas de leucémie congénitale, Bull. Soc. d'obst. et de gynéc. **11**:228, 1922.

17. Rhamy, B. W.: Leukemia in the Newborn, with Death at Birth from Traumatic Rupture of the Spleen, Am. J. Clin. Path. **8**:567, 1938.

blastosis foetalis or icterus gravis; if the pathologist is to call the disease congenital he should find more infiltration than could plausibly have occurred in the patient's extrauterine life. If he is to diagnose leukemia there should be infiltration with early cells rather than increase in activity of hemopoietic centers, and this infiltration should involve non-hemopoietic organs as well as the spleen, liver and lymph nodes. It will be seen that our case meets all of these requirements.

SUMMARY

A case of congenital leukemia has been reported. The literature has been reviewed, and some of the difficulties in diagnosis have been discussed. In 8 of the 9 cases reported which we have accepted as unquestionable instances of congenital leukemia the form was myeloid, and a single instance of probable congenital lymphatic leukemia has been found.

UNUSUAL FATAL PERFORATING WOUND OF THE CHEST

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The case to be described is deemed worthy of report because of the fatal outcome of what appeared at the time to be a minor superficial injury.

REPORT OF A CASE

R. M., a boy aged 4 years and 9 months, was riding on a tricycle along the sidewalk near his home when he fell over on his left side into a patch of recently cut weeds (fig. 1). He was observed getting up and a short while later falling again. The father, who was summoned, found the child unconscious and foaming at the mouth. He was immediately placed in the family car and rushed to the South Nassau Communities Hospital, not more than 2 miles (3.2 kilometers) from the scene of the accident. On arrival he was dead. The parents stated that the child had been allowed to go outdoors not more than fifteen minutes before the father was notified; so it was likely that not more than fifteen to twenty minutes had elapsed between the accident and death. Because of the unusual circumstances of the case, it was handled in the Medical Examiner's Office.

An autopsy performed fifteen hours after death showed marked anemia of the mucous membranes and a small amount of blood-tinged frothy fluid exuding from the mouth.

The only noteworthy feature seen on external examination was a lacerated wound in the left axillary fossa 7/8 inch (2.22 cm.) in length and 1/8 inch (0.32 cm.) wide. The periphery of the wound showed local hemorrhage for a distance of 1/8 inch on each side. The wound was located in the anterior axillary line just under the fold of the pectoralis major muscle and 1/2 inch (1.27 cm.) from its most superior portion (fig. 2).

When the thorax was opened, bloody fluid welled up along the line of incision of the left pleural sac. By measurement, the total volume of fluid and clotted blood within the left pleural cavity was found to be between 28 and 30 ounces (830 and 890 cc.). The left lung showed partial atelectasis of both lobes.

Inspection of the structures immediately beneath the cutaneous wound showed a perforating wound of the first intercostal space, with perforation of the lateral surface of the apical part of the parietal pleura just beneath. On closer inspection of the left lung, two linear lacerations were seen on the anterior lateral apical portion of the upper lobe. These were longitudinal and were separated from

From the Office of the Chief Medical Examiner of Nassau County.

each other by a bridge of lung tissue about ½ inch in width. Section along the tract of the wound showed macerated lung structure surrounded by hemorrhage which extended for ¼ inch around the edges of the tract. On section of the bronchi in the neighborhood of the tract, there was observed a small amount of blood-tinged mucus.

The thymus showed extensive hemorrhage into its subcapsular structure. The hemorrhage extended up into the neck along both carotid sheaths. There

Fig. 1.—Scene along the sidewalk showing stubble which remained after weeds had been cut with a scythe.

Fig. 2.—External wound on the left side of the chest.

was a small laceration in the capsule of the thymus, roughly oval in outline and measuring ¼ inch in diameter. This lay immediately over the left phrenic nerve.

Dissection of the structures of the supraclavicular and axillary region of the left side revealed no large bleeding point, the larger vessels in that area showing no injury.

Except for anemia and pallor of the organs and tissues, the remainder of the autopsy revealed nothing of note.

A reconstruction of the probable tract of injury showed an entrance wound in the anterior axillary line of the left axillary fossa just behind the fold of the

left pectoralis major muscle. It pierced the intercostal muscles between the first and the second rib, perforating the parietal pleura just beneath, and passed through the anterior lateral surface of the apex of the left lung, terminating in a lacera-

Fig. 3.—Photograph of stubble, showing sharp beveled ends.

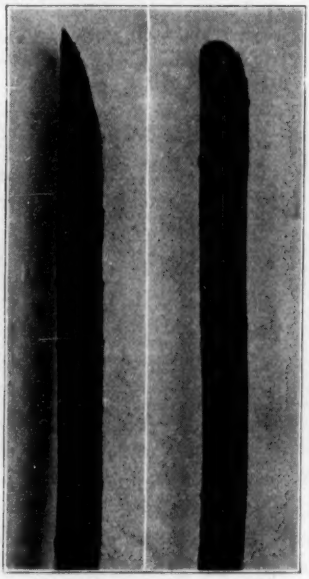

Fig. 4.—Close view of sharp, pointed ends of stubble.

tion of the capsule of the thymus. The greater portion of the bleeding probably came from the left superior intercostal vein or some small pericardial and thymic tributaries of the left innominate vein.

Subsequent examination of the scene of the accident revealed that an area 20 to 30 feet (6 to 9 meters) along the sidewalk recently had been cut with a

scythe. Stumps of elderberry bushes, 8 to 15 inches (20 to 38 cm.) in length and varying in diameter from ⅛ to ⅜ inch (0.32 to 0.95 cm.), had been left standing. The cut had been made on a bias, leaving sharp pointed ends (figs. 3 and 4).

COMMENT

The case is reported, first, because of the unusual nature of the accident, and second, because it shows that in many suburban communities a definite hazard to public health exists. This is due to the common method of cleaning brush by means of a scythe or sickle without taking the extra precaution of removing the remaining stubble.

Progress in Pediatrics

THE FRÖHLICH SYNDROME

REPORT OF THE ORIGINAL CASE

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The term "Fröhlich syndrome" is so frequently used as a diagnosis for obesity in childhood and adolescence that it has almost entirely lost the specific, well defined significance which it originally possessed. One reason for the vague and indiscriminate application of this diagnosis seems to be that many physicians are not familiar with the clinical picture which Fröhlich reported in 1901.[1] Since his paper is not readily accessible, the present report is written with the intention of reconstructing the clinical picture of this case. The original publication was given in the form of a lecture; most of the data were recorded in incomplete sentences. This style is not well suited to a verbatim translation. The present report follows the original description as closely as possible. The quotations from the older literature, which Fröhlich discussed in detail in his paper, have been omitted. The order in which the material was presented has been rearranged.

REPORT OF THE CASE DESCRIBED BY FRÖHLICH

R. D., a boy, was born in 1887. Information concerning the family history and the early development of the patient is not recorded. The child was apparently normal and healthy until the onset of the condition which brought him to the outpatient department of Dr. Nothnagel on Nov. 4, 1899.

The mother stated that since April the boy had been complaining of severe headaches about twice a week on coming home from school. Sometimes the interval between headaches was as long as two weeks. He had to go to bed, and he vomited about two hours later; occasionally he vomited immediately after he came home. The headaches were usually on the left; sometimes they were bilateral, being localized in the frontal region. The boy had continued to do good work at school; his memory was unimpaired, and he showed no signs of nervousness or hysteria. There was no history of previous disease or preceding trauma. His eyesight was good. Except for headaches and vomiting, he had no complaints. The functions of the bladder and the rectum were normal.

This study was aided by a grant from the Josiah Macy, Jr. Foundation.

From the Department of Pediatrics, College of Physicians and Surgeons, Columbia University.

1. Fröhlich, A.: Ein Fall von Tumor der Hypophysis cerebri ohne Akromegalie, Wien. klin. Rundschau **15**:883 and 906, 1901.

The physical examination failed to reveal any pathologic feature. Notwithstanding this, a detailed record was kept. The optic fundi presented no abnormality. They had been examined previously by Professor Königstein and had been found to be normal.

In view of the negative findings, it was felt that the patient suffered from hemicrania, and therapeutic advice was given accordingly.

On Aug. 19, 1901, he returned to the clinic with a number of serious complaints. The mother gave the following information: Since March 1899 the patient, who previously had been slim, had been rapidly gaining weight. In January 1901 he complained about diminishing eyesight on the left; not much attention was given to this complaint. In July the headaches recurred, and subsequently they increased in severity. The boy tired easily and vomited repeatedly, mostly after meals. Visual acuity diminished progressively until complete blindness in the left eye ensued. Later, vision in the right eye also began to fail.

On September 9 treatment with thyroid tablets was begun. After this, subjective improvement was noted. The boy had fewer headaches and did not complain of dizziness. He had not felt nauseated for ten days prior to the date on which this improvement was recorded. The appetite and sleep were good, but he lost some weight. His weight had been 54 Kg. (118.8 pounds) in August and was 51.5 Kg. (113.3 pounds) on September 23.

Results of the examination on this date were recorded in detail. Intelligence and speech appeared normal. All movements of the head were free. Only the left temporal region was sensitive to percussion. Examination of taste, smell and hearing gave normal results. There were no disturbances in the other cranial nerves except for the optic nerve. Motility and sensation of the extremities and of the trunk were entirely normal. The tendon reflexes, especially the patellar reflex, were active. No ankle clonus was observed; Romberg's phenomenon was not elicited. The function of the sphincters was normal. Examination of the visceral organs revealed no abnormalities. The urine was free from sugar and albumin.

The eyes were examined by Dr. Kuhn. The pupils were equal and 4 mm. in diameter. The left pupil did not react to light but reacted well in accommodation. The right pupil reacted immediately to light and in accommodation. Both eyeballs moved freely; no nystagmus was observed.

The left eye was amaurotic. The vision on the right was 5/20 (the measuring unit is not stated); it was not improved by glasses. There was right temporal hemianopia.

On general examination the patient, now 14 years old, appeared to be well developed and excessively well nourished (fig. 1). The body weight was 54 Kg. (118.8 pounds) in August 1901. The average weight of a boy of the same age and height was recorded by Fröhlich as 39 to 40 Kg. (The height of the patient is not recorded. From the average weight and the age of the patient, the height can be assessed as approximately 145 cm. [58 inches], which corresponds to the average height of a 13 year old boy. The excess of weight is approximately 36 per cent.) The adiposity had developed simultaneously with the appearance of the other symptoms. This chronologic connection was emphasized by Fröhlich. Under thyroid treatment the patient had lost 2.5 Kg. (5½ pounds) in three weeks, and as previously stated he weighed 51.5 Kg. (113.3 pounds) on September 23. The fingers, with the exception of the terminal phalanges, were plump; the hands appeared to be well padded. The bony structures did not in any way take part in the increase in volume. The largest accumulation of fat was in the sub-

cutaneous tissue of the trunk, particularly on the abdomen and in the neighborhood of the genitals. There the accumulation of fat was so enormous that it protruded markedly around the genitals. The penis, which was normally developed, appeared so deeply embedded in the fat tissue that the genitals approached the feminine type. The testes were small but were palpable in the depth of the masses of fat. Another considerable accumulation of fat tissue was observed in the mammary regions. A few small nodules could be felt in the mammary glands; no fluid could be expressed. Axillary hair was absent; at the genitals a few fine hairs were noted.

The hair of the skull was brittle, short and scanty; it had been falling out since the beginning of the disease. The skin was dry and scaling in many places. Over the trunk it could be lifted in thick folds together with the subcutaneous fat tissue. In other regions, especially over the fingers and wrists, the skin gave

Figure 1

Figure 2

Fig. 1.—The patient at 14 years of age. The plate is reproduced from Fröhlich's publication.[1]

Fig. 2.—The patient after operation; the age was not stated. The plate is reproduced from Biedl's book.[3]

the impression of being somewhat thickened. The nails had been growing rapidly; the patient stated that even though he cut them every three days, they grew to a considerable length during that period.

On the basis of the clinical course and findings, Fröhlich made the diagnosis of neoplasm of the hypophysis. The arguments by which he supported this diagnosis will be reported in the discussion.

The patient was demonstrated in Vienna at a meeting of the Society for Psychiatry and Neurology on October 12. In a footnote of the publication, a subsequent examination of the eyes is recorded. On November 12 the inner half

of the right papilla showed marked reddening; it was opaque and slightly swollen. The diagnosis of neuritis nervi optici was made.

Information concerning the subsequent course is recorded in several publications of von Eiselsberg and von Frankl-Hochwart [2]; Fröhlich was clinical assistant of the latter at the time when he examined the patient and reported the case.

After treatment with thyroid, the headaches diminished, and the vision in the right eye improved; it appeared to be normal on Jan. 27, 1903. The accumulation of fat did not change significantly. The patient did not develop sexually and had no erections. After September 1905 his general condition grew worse. In January 1907 hemianopia was again observed. A roentgenogram of the skull was obtained and interpreted by Dr. Holzknecht. The body of the sphenoid and the dorsum sellae were destroyed; the anterior clinoid processes were preserved. Examination of the eyes on June 15, 1907, showed genuine atrophy of the optic disk on the left; the temporal half of the right papilla was atrophic. The patient could count fingers at a distance of 2.5 meters. Glasses did not improve the vision of the right eye. The left eye was amaurotic. The right visual field showed temporal hemianopia. The 20 year old patient was of average stature and weighed 65.2 Kg. (144 pounds). His general appearance had remained infantile feminine.

Since the patient suffered from severe headaches and his eyesight was rapidly decreasing, an operative procedure appeared justified. The operation by the nasal route was performed by von Eiselsberg on June 21. In the depth of the sphenoid sinus the whitish membrane of a cyst the size of a hazelnut was encountered. After incision in the midline, several spoonfuls of a fluid resembling old blood drained out. By measuring with the finger and comparison with the roentgenogram, it could be ascertained that the cyst which contained this fluid corresponded to the hypophysis. The walls of the cavity were cut away as far as could be done without damaging the optic chiasm and the carotid arteries.

Histologic examination of the removed tissue by Dr. Stoerk suggested that it might be a carcinoma or, more probably, an adenoma of the hypophysis in the precancerous stage.

The postoperative course was favorable. For a brief initial period it was accompanied by elevation of the temperature. The wound was essentially healed on the twelfth day. The removal of the anterior wall of the frontal sinus resulted in a disfiguring flattening of the glabella, which was conspicuous (fig. 2).

There was considerable improvement in the general condition. The headaches became less frequent, and the eyesight improved. On July 27 vision on the right was 1/35; the visual field of this eye had considerably enlarged.

The further treatment consisted of dietary measures, hydrotherapy and galvanotherapy. During August occasional headache and vomiting occurred; in the interval the patient felt completely well.

Examination of the eyes on September 7 showed further improvement. The vision was 2/35; the temporal half of the visual field appeared further extended. Curiously, in the left eye, which was totally amaurotic, there was slight but distinct reaction of the pupil to light.

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2. von Eiselsberg, A., and von Frankl-Hochwart, L.: Ueber operative Behandlung der Tumoren der Hypophysisgegend, Neurol. Centralbl. **26**:994, 1907; Ueber operative Behandlung der Hypophysistumoren, Wien. klin. Wchnschr. **20**: 1341, 1907; Ein neuer Fall von Hypophysisoperation bei Degeneratio adiposogenitalis, ibid. **21**:1115, 1908.

At a general examination on September 8 the patient stated that he had had no significant complaints since August 31. He had lost 2 Kg. (4.4 pounds) during the last six weeks.

The patient continued to feel well, and the improvement of his eyesight persisted. At an examination on July 11, 1908, he stated that he had not suffered from headaches and dizziness for several months. He had been working as a representative of a factory. Since March 1908 erections had occurred; there was some growth of hair in the axilla, in the pubic region and on the wrists. The body weight was approximately 70 Kg. (154 pounds).

Biedl [3] reported on this patient in 1913. At that time, the general improvement had persisted for several years. After the initial progress, no further change had occurred. The patient continued to be of infantile habitus and showed no significant decrease in his deposits of fat.

(No symptoms which might be interpreted as indicative of diabetes insipidus were mentioned in any of the reports.)

DISCUSSION WHICH FOLLOWED FRÖHLICH'S REPORT

Fröhlich described in great detail how he had arrived at the diagnosis of tumor of the hypophysis. The onset with headaches and vomiting and the slow course pointed to a gradually progressive, expanding process localized at the base of the skull, more specifically in the region of the optic chiasm. The unequal involvement of the eyes led him to the assumption of a neoplasm in the region of the hypophysis. He was, however, not satisfied with a purely topical diagnosis. He thought first of a tumor of the hypophysis in connection with myxedema. But the absence of characteristic changes of the skin, the normal intelligence and the presence of a palpable thyroid gland militated against this diagnosis.

Reviewing the older literature on tumors of the hypophysis, he cited numerous reports in which trophic disturbances other than acromegaly had been recorded. Most frequently, adiposity and the feminine infantile appearance had been prominent symptoms. None of the older authors had recognized the diagnostic importance of these associated symptoms. Thus far, only fully developed acromegaly with involvement of the face and head in connection with local cerebral symptoms had justified the diagnosis of pituitary disease.

It is to the great clinical merit of Fröhlich that he recognized for the first time the diagnostic significance of adiposity and other trophic disturbances, such as genital underdevelopment, for the localization of pathologic cerebral processes. Fröhlich concluded that these disturbances point to the hypophysis itself as the origin of the neoplasm.

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3. Biedl, A.: Innere Sekretion: Ihre physiologischen Grundlagen und ihre Bedeutung für die Pathologie, ed. 2, Berlin, Urban & Schwarzenberg, 1913, vol. 2, p. 178.

However, it appears now that his first assumption of "region of the hypophysis" was more correct. No attempt will be made to review the whole extensive literature on this controversial subject. Only a few of the more important contributions will be mentioned. In 1904, Erdheim [4] took issue with Fröhlich's conclusions. He agreed with Fröhlich that adiposity occurs frequently in the presence of tumors of the hypophysis. However, he doubted that the hypophysis in its role as an endocrine gland is responsible for the development of obesity. On the basis of autopsies, he showed that only those tumors of the hypophysis which grow beyond the sella turcica are associated with obesity. In addition, adiposity had been observed not only with tumors of the hypophysis but with neoplasms originating in, or near, the infundibulum without involvement of the hypophysis. He postulated that the symptom of adiposity should be interpreted with the greatest reserve; if its presence was used as a diagnostic aid in the localization of a cerebral process, it pointed to a lesion at the base of the brain. Erdheim discussed this problem again in 1916 [5] and stressed the conception that adiposity cannot be explained as due to a lesion of the hypophysis. He considered retardation in growth and sexual underdevelopment, however, as symptoms of a specific lesion of the anterior lobe of the hypophysis.

Bartels,[6] who coined the expression "dystrophia adiposogenitalis," which is frequently used as a synonym for "Fröhlich syndrome," expressed himself in a similar way. Von Eiselsberg and von Frankl-Hochwart,[2] who gave full credit to the diagnostic importance of Fröhlich's formulation, considered the newer conclusions of Erdheim and Bartels to be more correct.

Among the more recent clinical and pathologic publications, mention will be made only of Fulton and Bailey's [7] report concerning tumors in the region of the third ventricle. These authors considered adiposity as a part of the "hypothalamic syndrome," which may also include hypersomnia, polyuria, genital dystrophy and perhaps glycosuria. They considered it extremely doubtful that the hypophysis plays an essential role in the production of adiposity.

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4. Erdheim, J.: Ueber Hypophysenganggeschwülste und Hirncholesteatome, Sitzungsb. d. Akad. d. Wissensch. Math.- naturw. Kl. **113**:537, 1904.

5. Erdheim, J.: Nanosomia pituitaria, Beitr. z. path. Anat. u. z. allg. Path. **62**:302, 1916.

6. Bartels, M.: Ueber Plattengeschwülste der Hypophysengegend (des Infundibulums), Ztschr. f. Augenh. **16**:407 and 530, 1906.

7. Fulton, J. F., and Bailey, P.: Tumors in the Region of the Third Ventricle: Their Diagnosis and Relation to Pathological Sleep, J. Nerv. & Ment. Dis. **69**: 1, 145 and 261, 1929.

The information obtained in animal experiments became of value only after technics had been developed which permitted the production of separate lesions in the hypophysis and in the adjoining region. The experiments of Bailey and Bremer [8] were the first to prove that a lesion of the hypothalamus alone results in adiposogenitalism. Numerous reports have since confirmed this observation. Although the evidence concerning the cause of adiposity is not yet clear, there is no experimental support for the assumption that adiposity can result from a lesion of the hypophysis alone. In addition hypophysial extracts fail to cure adiposity resulting from a hypothalamic lesion.

For a long time it seemed that the conflicting theories could be reconciled by the assumption that an intimate anatomic and physiologic connection existed between the hypophysis and the surrounding nerve centers. Recent anatomic investigations [9] have shown this concept to be false. There are no veins of any consequence passing from the infundibular stem to the hypothalamic region or vice versa.

COMMENT

The use of the term "Fröhlich syndrome," with its implication of hypopituitarism, for juvenile obesity was a harmless misconcept as long as the resulting therapeutic activity consisted of nothing more than the oral administration of inefficient hypophysial preparations. In recent years, however, highly potent pituitary extracts have become available; their indiscriminate use cannot be defended on the basis of an incorrect diagnosis.

Finally, it must be pointed out that the majority of obese children do not even conform in their general appearance to the description of Fröhlich's patient. They grow and develop at a rate faster than that of normal children.[10] This observation is in direct contrast to the physical and sexual infantilism which is an essential part of the Fröhlich syndrome. To label obese children as representatives of the Fröhlich syndrome and to consider them as suffering from hypopituitarism involves a twofold error in diagnosis.

SUMMARY

The original case described by Fröhlich in 1901 has been presented in English. The "Fröhlich syndrome" consists of trophic disturbances,

8. Bailey, P., and Bremer, F.: Experimental Diabetes Insipidus, Arch. Int. Med. **28**:773 (Dec.) 1921.

9. Wislocki, G. B., and King, L. S.: The Permeability of the Hypophysis and Hypothalamus to Vital Dyes, with a Study of the Hypophyseal Vascular Supply, Am. J. Anat. **58**:421, 1936.

10. Bruch, H.: Obesity in Childhood: I. Physical Growth and Development of Obese Children, Am. J. Dis. Child. **58**:457 (Sept.) 1939.

such as infantilism, genital dystrophy and adiposity, in the presence of a neoplasm at the base of the brain.

The assumption of Fröhlich that these trophic disturbances indicate that the tumor originates from the hypophysis itself has not been borne out by subsequent clinical and experimental evidence.

This statement does not detract from the value and significance which Fröhlich's conclusions had at the time when they were published. The term "Fröhlich syndrome" deserves to be remembered as a historical concept; however, it is no longer correct in its original interpretation and has outlived its clinical usefulness.

THUMB AND FINGER SUCKING IN CHILDHOOD

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NEW YORK

Thumb and finger sucking are of importance because of the concern they cause in parents. The pediatrician to whom the alarmed parents come for advice is often as confused about the significance of the practice as are the parents themselves. He finds it difficult to help them or to allay their fears. In order to do this successfully he is in need of a working knowledge of the significance of the habit at different age levels. He needs to know what harm, if any, may result; what factors lead to the development of the habit, and what measures should, or should not, be taken to handle the situation adequately.

Sucking of the thumb or the fingers occurs at some time in nearly every child's life. Sucking of the toes occurs less frequently but is not rare. Many young infants suck their fingers only when hungry; others indulge in the practice for its pleasurable sensations. Associated movements,[1] such as pulling at an ear, twisting the hair, rubbing some soft fabric, or handling of the genitals or nipple, are sometimes found. Some of these have been said by some investigators to be residuals of the fondling of the mother during nursing. The accessory movement may be present without finger sucking. I recall a child who persisted in the associated movement of pulling out her hair after the thumb sucking had been given up. In some children the associated movement may be brought into the sucking when the finger is left out, as in the sucking of woolly objects or the rubbing of them on the lips. The approach to these other sucking habits is the same as it is to finger sucking, and the subsequent remarks may be regarded as applying to these as well.

Concern of parents regarding thumb and finger sucking in their children is largely a result of the scares about the harm done to the

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1. (*a*) Levy, D. M.: Fingersucking and Accessory Movements in Early Infancy: An Etiological Study, Am. J. Psychiat. **7**:881, 1928. (*b*) Lindner, S. L.: Das Saugen an den Fingern, Lippen, u. s. w. bei. den Kindern, Jahrb. f. Kinderh. **14**:68, 1879.

child by the habit. This has come about since the beginning of the present century as a result of the warnings of alarmists. But if the habit is so harmful, why is the alarm so recent? Kanner,[2] an assiduous student of folklore, made the following interesting comment:

> Even folklore, seizing so promptly upon every portion and function of the human body, attributing great significance to such things as sneezing and yawning and firmly believing in the worm etiology of nose picking or grinding of teeth, has failed to consider finger sucking as worthy of its attention.

Much of the alarm caused in parents is about the effects of finger sucking on the conformation of the palate and the dental arches. This has led to a struggle to stop the "pernicious habit" as soon as the child shows any tendency to place his thumb or fingers in the mouth. Many mechanical restraints have been devised which keep the child from being able to get his hands near his mouth, and others, attached to the teeth, make the act decidedly uncomfortable.[3] Drugs are painted on the fingers, or a program of constant nagging and bickering is set up, which usually serves only to fix the child's attention on the act. At best, these devices produce only a temporary effect. They are most apt to work during the infancy period and are definitely contraindicated after the first year. A sober viewing of the facts concerned with the deformities of the mouth resulting from finger sucking shows no cause for alarm and certainly no reason for inconveniencing the child or his parents with any such program for eliminating opportunities to suck. To be sure, a malocclusion may and can be produced by finger or thumb sucking. It is characteristic[4] and usually occurs when the finger is sucked with the volar surface upward, producing pressure on the palate

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2. Kanner, L.: Some Pediatric Problems of Behavior in Infancy and Early Childhood, J. Pediat. **13**:417, 1938.

3. Anderson, W. W.: Thumb-Sucking Apparatus, Am. J. Dis. Child. **39**: 70 (Jan.) 1930; **46**:90 (July) 1933. Heimlich, A. F.: Correction for Thumb- and Finger-Sucking Habits, Internat. J. Orthodontia **21**:1138, 1935. Swinehart, E. W.: Structural and Nervous Effects of Thumbsucking, J. Am. Dent. A. **25**: 736, 1938. Van der Bogert, F.: Device for the Control of Hands in Facial Eczema and Thumbsucking, Arch. Pediat. **51**:810, 1934.

4. (*a*) Bonney, T. C.: Facial Deformities: The Results of Thumb-Sucking, Tongue- and Lip-Biting, Mouth Health Quart. **4**:12, 1935. (*b*) Chandler, T. H.: Thumbsucking in Childhood as a Cause of Subsequent Irregularity of the Teeth, Boston M. & S. J. **99**:204, 1878. (*c*) Johnson, L. R.: Habits and Their Control in Relation to Malocclusion in Children, J. Am. Dent. A. **24**:1409, 1937. (*d*) McCoy, J. D.: Applied Orthodontia, ed. 3, Philadelphia, Lea & Febiger, 1931. (*e*) Pullen, H. A.: Abnormal Habits and Their Relation to Malocclusion and Facial Deformity, Internat. J. Orthodontia **13**:233, 1927. (*f*) Rowlett, A. E.: Some Observations on the Prevalence of Excessive Overbite and Its Possible Etiological Significance, ibid. **9**:668, 1923.

and pushing the upper incisors forward. Lewis,[5] in a series of papers, has shown that if the sucking is stopped before the eruption of the permanent teeth, the deformity will spontaneously cure itself. Thus it can be seen that sucking during the early years produces no lasting or serious deformities. Unfortunately, other papers in the dental literature, holding an opposite view of prognosis, usually speculative in nature and not based on sound scientific observation, are more frequent and better known.

The alarmists, not content with limiting themselves to local damage to the teeth and palate as a result of sucking habits, have extended the ill effects to include deformities of the fingers and scoliosis, and have even suggested that the habit may be a contributing cause[6] of diseased tonsils and adenoids. No valid evidence has been offered to prove any of these suggestions. Dental caries has been attributed to the resulting malocclusion; one writer[6b] went even further and suggested that the early loss of teeth as a result of caries, leading to digestive disturbances in adult life, is a more remote result of this "innocent habit." Digestive disturbances in infancy have also been attributed to the sucking. Colic has been said to be a direct result of the swallowed air, and the thumb, has been said to be a carrier of disease bacteria into the mouth and digestive system.[6a] However, no evidence has been adduced that thumb suckers are any more susceptible to disorders of the gastrointestinal tract than are other children.[7a] It has also been said that the sucking act will cause changes in the facial expression which markedly affect future character development.[6b] This statement seems to be merely another alarm which has been broadcast in 3 inch headlines. On the other hand, there is some evidence to show that the child who has been shamed, teased or nagged about his sucking may be pushed into a solitary, unsocial type of behavior, which may markedly affect his future adjustment.[7b]

Thus it can easily be seen that the widespread alarm about the ill effects is needless in early childhood. The local damage to the mouth is temporary if the habit is discontinued before the eruption of the permanent teeth, or by the age of 6 years. The other bad effects exist largely only in the imaginations of their proponents.

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5. Lewis, S. J.: Thumbsucking: Cause of Malocclusion in Deciduous Teeth, J. Am. Dent. A. **17**:1060, 1930; Undesirable Habits Influencing the Deciduous Dentition, ibid. **18**:1766, 1931; The Effect of Thumb and Finger Sucking on the Primary Teeth and Dental Arches, Child Development **8**:93, 1937.

6. (*a*) Watson, J. B.: Psychological Care of Infant and Child, New York, W. W. Norton & Company, Inc., 1928, p. 133. (*b*) Woodcock, I. S.: Thumbsucking, M. Rec. **139**:328, 1934. (*c*) Lindner.[1b]

7. (*a*) Bakwin, R. M., and Bakwin, H.: Psychologic Care During Infancy, J. Pediat. **12**:71, 1938. (*b*) Kanner, L.: Child Psychiatry, Springfield, Ill., Charles C. Thomas, Publisher, 1935, p. 331.

There is yet another cause for alarm in parents. Thumb and finger sucking have been characterized as a form of "larval masturbation" [8] and as an "exquisitely sexual act." Some writers have stated that the cause of much of the unnecessary alarm in parents is that they unconsciously realize this connection, and their own early conflict about masturbation is brought to the surface.[9] Sucking is, to be sure, an exquisitely infantile pleasurable reaction, but certainly not sexual in the usual sense of the word as applied to adult sexual reactions. One would feel that the child who needs to use so purely an infantile means of getting satisfaction is failing to obtain sufficient satisfaction out of life, and it behooves one to look for the sources of dissatisfaction. The pleasurable component cannot be denied, nor can the factor of some lack of self control.

Clinically it is useful to consider this symptom from the point of view of the age of the child. The significance of the act seems definitely to vary with the age of the child. It is convenient to divide children into groups according to their age level: the infant, the runabout or preschool child and the older child. The following remarks will tend to overlap in some of the groups, but in general the division will hold quite satisfactorily.

The newborn infant will vigorously suck on almost any object that is placed in contact with his lips. This persists as a more or less purely reflex activity for several months. In the neonatal period the infant will suck for as long as forty-five minutes on a dry nipple before he gets tired, and then, apparently as a result of fatigue, he will refuse to take the formula which is offered to him. Later, by the fourth or fifth month, he will not suck as long or as vigorously unless he gets something for his efforts. He will, however, suck at his fingers when hungry or when, after feeding, his appetite has not been satisfied. Sucking also occurs when the fingers reach the mouth in the ordinary course of manipulations of the arms. This occurs frequently in the neonatal period and then disappears until about the fourth month, either as a result of changes in the body structures which prevent easy access to the mouth [10] or as a result of the abolition of subcortically initiated movements of the arms, followed by the emergence of cortical control. At

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8. Freud, S.: Three Contributions to the Theory of Sex, translated by A. A. Brill, New York, Nervous and Mental Disease Publishing Company, 1925, p. 42. Isaacs, S.: Bad Habits, Internat. J. Psycho-Analysis **16**:446, 1935. Schmideberg, M.: "Bad Habits" in Childhood: Their Importance in Development, ibid. **16**: 455, 1935.

9. English, O. S., and Pearson, G. H. J.: Common Neuroses of Children and Adults, New York, W. W. Norton & Company, Inc. 1937, p. 96.

10. Gesell, A., and Ilg, F. L.: Feeding Behavior of Infants, Philadelphia, J. B. Lippincott Company, 1937.

any rate, the infant will begin to suck the fingers when the hand comes in his mouth. It would seem that the reasonably contented and well fed infant does not tend to suck to the exclusion of his other activities. This "near-physiologic" type of pleasurable sucking is at its peak during the so-called hand to mouth reaction period, between the fourth and the tenth month of life. The child uses the tactile sensation in his lips and mouth as a rather definite means of exploring his environment. All are aware of the tendency among infants of this age to pick up an object, look at it, mouth it, look at it again and then repeat the process. The tendency to mouth is replaced by the use of the tactile sensation in the fingers as the hand to mouth reaction gradually disappears. Gesell [10] has noted that the tendency to mouth is most marked during the periods of teething and will almost completely disappear in between teething periods. A great many children will have a recrudescence of the hand to mouth reaction with the attendant sucking behavior during the teething period of the fourteenth or fifteenth month.

It is of interest to mention here the work of D. M. Levy, who has published several interesting studies on the causation [11] of thumb and finger sucking, which have been summarized in a recent paper by Kanner.[2]

Levy has in an interesting study come to the conclusion that in a majority of instances it is due to insufficient lip movements or incompleteness of the sucking phase of the feeding act, regardless of the type of feeding (breast, bottle or mixed). He named as the most frequent causative factors: (1) spontaneous withdrawal from a too rapidly flowing breast or bottle; (2) forced withdrawal from sucking at the termination of too short a period of time; (3) changing feeding schedules with increasing intervals between feedings and diminished duration of the feeding time. Each nursing period, according to Levy, begins with the child tense and restless because of hunger. As the feeding continues, he becomes relaxed, and after a fully satisfying meal he is sufficiently relaxed to fall asleep. If, however, he is not fully satiated, he may suck his thumb or fingers, thus maintaining something of the pleasures of nursing until he falls asleep. There can be nothing seriously undesirable about this form of behavior, provided it does not become chronic. But if the feeding is not entirely satisfactory, there is danger that each meal will so frequently be followed by thumb-sucking that the act will become thoroughly habitual. Levy subsequently extended his investigations to dogs, calves, and chickens. If the feeding act was disrupted before the point of satiety was reached, a sucking habit resulted in dogs in the form of licking of the paw or the body of another dog, besides sucking of various inanimate objects. In calves, the licking habits concerned the ears of other calves, painted boards, clothes, anything suitable for licking. The pecking behavior of chickens, raised

11. (*a*) Levy, D. M.: Experiments on Sucking Reflex and Social Behavior of Dogs, Am. J. Orthopsychiat. **4**:203, 1934; A Note on Pecking in Chickens, Psychoanalyt. Quart. **4**:612, 1935; Thumb or Finger Sucking from the Psychiatric Angle, Child Development **8**:99, 1937; Angle Orthodontist **7**:100, 1937; On Instinct Satiation: An Experiment on Pecking Behavior of Chickens, J. Gen. Psychol. **18**:327, 1938. Levy.[1a]

on a platform of wire mesh (and that of turkeys) was found to follow the same law of instinct satiation as in the studies of sucking behavior. Levy stated that the percentage of finger-suckers was highest in infants fed every four hours, less in a group fed every three hours, and least in a group fed irregularly.

Gesell,[10] on the other hand, concluded from his own observations of young infants that there are so many exceptions to this that it cannot be laid down as a general factor. It would also seem that there are large individual variations in the sucking needs among different children. Some apparently well fed and happy children are able to give up the bottle at 5 or 6 months without developing any abnormal sucking responses, whereas other, equally well fed, happy, and contented babies are unwilling to give up the sucking experiences until well after the first birthday.

For the ordinary, transitory, so-called "near-physiological" form of finger sucking, no therapeutic plans are necessary, aside from reassuring the parents that all is well.[12] In dealing with an infant whose sucking is more marked and occupies a good deal of his waking time, the thumb sucking is to be regarded as a symptom that he is not getting all of the satisfaction out of life that he needs. A change in the feeding schedule or arranging for him to get a little cuddling or "mothering" is usually all that is needed.[13] It is to be noted that the technic of feeding is just as important from the infant's point of view as is the amount given. Many mothers are prone to place the child in his crib or carriage and prop up the bottle on pillows after the child is a few months old. This is apt to be regarded by the child as emotional deprivation, resulting from a withdrawal of the mother. In other instances, forcing more food than the child needs or insisting that he take foods which obviously upset him are the sources of an unpleasant and unsatisfying feeding situation.

In the latter part of the first year and throughout the next three or four years of life, finger sucking may be observed in various situations.[14] When the child is bored, he usually begins to derive his pleasures from sucking habits; but the sucking does not become a problem unless he is chronically deprived of adequate amusement. This does not mean, it should be stressed, that he should be constantly amused and played with by some adult member of the family group. However, it is surprising how many parents will leave their young child with not one or only one toy, expecting him to play for considerable periods of time, but forgetting that as yet his attention span is brief and that he is incapable of keeping at one type of play activity for more than a brief period. Gesell [10] observed an interesting contrast in the behavior of children

12. English and Pearson.[9] Kanner.[2]

13. Aldrich, C. A., and Aldrich, M. M.: Babies Are Human Beings, New York, The Macmillan Company, 1939, p. 100.

14. English and Pearson.[9] Gesell and Ilg.[10] Kanner.[7b] Kanner.[2]

who, during their second year, were being trained in toilet habits by somewhat overzealous mothers. When these children had no desire to defecate they would begin to suck their fingers, whereas if they actually needed to use the toilet at that time there would be no sucking.

The child is apt to suck when he has been scolded or punished. Frustration brings on a sucking response; the child turns to the pleasures of thumb and finger sucking or to one of the other related sucking habits in times of stress, when his other satisfactions in life are at a minimum. The active three or four year old who is constantly being nagged at because of his efforts to satisfy his strong sense of curiosity and to explore all possible nooks and crannies of his immediate environment frequently responds to the nagging with sucking. Likewise, a young child living in a small city apartment without any adequate outlets for his motor activity and boisterous play will react in a similar manner. This period of life is one which produces in the child many frustrations. When he enters the third year, the so-called period of resistance or negativism [15] is apt to have started. This is a difficult period for parents to handle because of the constant, "No, no," and "I won't," from the child. Two important factors in the development of this resistive behavior are frustrating to the child. It occurs when the withdrawal of the mother from the care of the child is at its peak. The child is now expected to feed himself, to make his toilet wants known, and to stand on his own feet and use his own resources to keep himself occupied more than before. This means that the mother has a less active part in doing things for him, and he does not like it. In addition, it is during the third year that many neuromotor patterns in the child are maturing at different rates of speed. The child is able to start an act but is not able to complete it. McGraw [16] has remarked on the similarity of this to the period when the child first learns to stand. He cannot sit down, so he cries; but as soon as he is put down, he stands up again. The increased irritability during the third year is in part attributable to this constant frustration. His desires are beyond his capacity to complete them because of the imperfections of his tools. All have observed how angry a young child can get when he tries with his as yet imperfect language to explain something, particularly when he cannot make his hearers understand what he means. These frustrating experiences are to a certain extent unavoidable, and occasional short-lived sucking episodes as a response are the rule rather than the exception. It is only when more of a burden is thrown on the child than he should reasonably be expected to bear that the sucking response becomes so habitual that

15. Thom, D. A.: Everyday Problems of the Everyday Child, New York, D. Appleton-Century Company, Inc., 1934., p. 104. Kanner.[2]

16. McGraw, M. B.: Later Development of Children Specially Trained During Infancy, Child Development **10**:1, 1939.

the child prefers to indulge in it to the exclusion of other more socially acceptable behavior. It is seen in those children who because of poor handling with misunderstanding on the part of the parent or nurse are given little opportunity to gain normally satisfying emotional experiences.

I should like to quote briefly from the history of a child who was brought to me because of a tendency to suck woolly objects. She would do this at every opportunity and while doing it could seem so completely entranced that she was unaware of what was going on about her. She had been adopted at the age of 3 months; at that time she was rather a puny child who looked as if the world had not given her a fair deal. This plaintiveness appealed to the adoptive parents who, however, fearing that perhaps they had adopted a child who might not measure up well in the particular social sphere in which they moved, laid down a program for the systematic training of the infant. The abnormal sucking behavior began at about 2 years of age when instead of the former motherly nurse, who fairly exuded kindness, a rather prim, tense, perfectionistic nurse came, who was determined that this child should conform in every respect to the patterns laid down for her. The child, for instance, was expected at 28 months of age to keep up a sustained interest in a garden which she was helped to plant. The nurse was more interested that she hold the crayon correctly than that she get satisfaction from scribbling. Similarly, she was taught to play golf at about 30 months of age, with the accent on the correctness of her swing rather than any fun she might get out of hitting the ball. The child did get some satisfaction out of the concern that her sucking behavior caused in those about her, but did most of her sucking when alone or when not aware that she was being observed. The sucking of wooly objects disappeared when a program which allowed for satisfactions commensurate to her age, which was then 4, was arranged. This, of course, called for the elimination of the nurse.

The tired or physically ill child will tend to suck his thumb or fingers. The proper treatment in the first instance is to eliminate the causes of the chronic fatigue. Most parents are aware of the tendency among children of this age to suck their fingers when they are tired and the bedtime hour draws near. In children of all ages there is a tendency for babyish behavior to occur during a time of acute physical illness. If the convalescence is wisely handled and the child is not left unhappy and discontented, the symptom will usually disappear with the return to normal activity.

Treatment of this symptom during the 2 to 5 year period, it will be seen, consists of alleviating the more obvious causes of emotional dissatisfaction in the child. Occasional sucking is the rule in children of this age and is not to be viewed with alarm, since even if a deformity is produced it will cure itself if the sucking disappears before the permanent teeth erupt. Restraints are contraindicated at this age.[17] They start a power contest in which the child usually wins, and, in addition, they serve to fix the child's attention on his symptom. A direct attack on the symptom is not needed. Very satisfactory results are

17. English and Pearson.[9] Kanner.[7b]

obtained without enlisting the child's interest in abolishing the symptom. In addition, it is frustrating to the child when he is unable to break himself of the habit because of the persistence of the underlying causes. When the underlying dissatisfactions are done away with, there ceases to be any reason for the symptom, and it disappears. When the thumb or finger sucking has become the battleground over which the struggle between parent and child takes place, of course any direct attention to the symptom is fruitless. It should be remembered that attention getting is frequently only a small part of the picture, and the physician who limits his approach to the problem with this formulation will not get very satisfactory therapeutic results.

Thumb sucking in children above the age of 5 or 6 years is not to be viewed as casually as in younger children.[17] With older children the danger of lasting deformity is greater. In addition, the symptom itself is, in the vast majority of cases, not on such a simple reactive basis. Here we see it as a part manifestation of a more general emotional and social immaturity which, unless measures are taken, may lead to a serious and lasting personality disorder. The emotional and social immaturity of these children of school age may arise out of a variety of underlying situations.[18] The protected child of indulgent and oversolicitous parents is ill prepared for the give and take life with his schoolmates. His social development has been retarded by his handling, and his lack of success in dealing with the social relationships at school adds another difficulty to those with which he is already struggling. In other children the retardation of social and emotional development seems to represent a regression to more infantile behavior patterns when the child meets with a difficult or intolerable life situation. Most of the children of this age are teased by their playmates because of their finger sucking; they are frequently regarded as being "sissies" by their schoolmates, intolerant of difference, as are all children of this age. The attention of the child then becomes centered on the sucking; often he cannot stop, no matter how hard he tries, because of the persistence of the underlying factors. He is apt to develop a solitary, unsocial type of behavior, which makes his problems even more difficult to unravel. I shall not discuss all of the ramifications in the development of the symptom in this age group, as it will vary in different children. Adequate handling involves a broad program of investigation and personality and environmental readjustment. The symptom of finger sucking occurs as only one of a group of traits which the child exhibits, and the attack must be made at the more general underlying causes. I shall limit my discussion of the symptom in this age group to citing 2 cases in which, among other symptoms, thumb or finger sucking occurred.

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18. Kanner.[7b] Thom.[15]

A 5½ year old girl, whose name was Catherine, was brought to the clinic with the story that since the birth of a younger brother, two and a half years before, she had sucked her fingers and wet her bed. Previously, she had been free from enuresis since the age of 14 months. The boy was the obvious favorite of the mother and was whole-heartedly resented by the patient, who did not hide the fact that she considered him a nuisance. Catherine was a discontented, irritable child who made large demands on her mother's time and was apt to respond with a tantrum when she felt that she was not getting a fair share of her mother's attention. She had not as yet gone to kindergarten because, although repeated attempts to vaccinate her had been made, a take had not as yet been obtained, and the school would not accept her without a vaccination certificate. The child did not seem to be particularly bothered because she could not go to school. When seen, she was observed to have a baby talk type of speech difficulty, the onset of which the mother did not remember. She did recall, however, that the child had spoken very clearly at the age of 2. Catherine did not much care whether she grew up or not. She stated that she would like to be younger than her brother and that babies had much more fun out of life. The mother was helped to rearrange her time at home so that Catherine received a fair share of her attention and no longer was made to feel less important than her brother. The mother, it seemed, preferred the brother because of Catherine's irritability. It was explained to her that as Catherine's difficulties cleared up the general behavior would improve. Catherine, in turn, was aided in developing a more normal attitude toward her brother through a discussion of her problem. She was also helped to gain more satisfaction out of the growing-up process. Kindergarten was held out to her as a fun-giving experience. Admission was arranged after an intradermal vaccination had been done. The family bought her a bed at home to take the place of the crib in which she had been sleeping. It is noteworthy that the enuresis stopped the first night she slept in her new bed. Within two months all the symptoms had cleared up. Catherine was a happy, contented girl who helped her mother about the home with the housework and who when she now came to the clinic stated with conviction that when she grew up she wanted to be a "big lady typewriter."

Another child, a girl of 8 years, was brought to the clinic because of the development of fears, six or eight months before. (It is of no significance that both of these patients are girls.) The child had gradually become fearful of meeting strangers and would not sleep alone at night because she was afraid that some one might come in and rob the house. She had terrifying dreams of kidnapers and that the home was burning down. For the past year she had been wetting her bed every night, although she had previously been free from enuresis since the age of 2. The mother complained that the child had been "acting like a baby" for a slightly shorter length of time. On further inquiry it was learned that by this she meant a mild tendency to baby talk, an increasing desire to be spoon fed by the mother, thumb sucking which, while not intense, did impress the mother as a problem, and a tendency to cry at the slightest provocation. Up to about a year before, the child had been considered by the parents as being perfectly normal. Eighteen months before, the father had been seriously ill with pneumonia, which left him with a chronic pulmonary infection, finally diagnosed as bronchiectasis. He had been a subway worker. When, after having been ill a year, he was finally permitted to return to work, it was with the proviso that he not be under ground. There was considerable worry in the home over the financial status. The family asked very little from life; the mother referred

to the days when the father was working as the "good old days when we had money" (he had made $22 a week). They had been upset at the prospect of "going on relief" and at losing their financial independence. During the months of the father's illness and unemployment they had gradually spent all of their savings, sold some of their furniture, and borrowed from relatives in order to postpone the day when they would become public charges. Both of the parents were upset that the father might never be able to become a wage earner again. All of this worry in the home about health and finances was reflected in the child. The two younger children were not referred to us because they had not been observed to have fears by the pediatric clinic physicians. Both, however, were as much upset as was their sister. The mother did not spontaneously volunteer much of the trouble at home, as she feared being pitied. It was obviously too much to expect of the child to adjust satisfactorily to this home situation. Finally, the mother was referred to a family case work social agency, which has been aiding them financially without causing the stigma of "being on relief" to be applied to them, and this is helping the family to face the truly difficult problems with which they are confronted. The improvement in the children has been slow and intermittent.

It can be easily seen that with these two children any direct attack on the thumb sucking would have been to no avail. The importance of the sucking becomes lost in the more complex pattern of the child's reactions.

In conclusion, during infancy, thumb and finger sucking occurs as a natural result of the hand to mouth reaction, which is most prevalent from the fourth to the tenth month of life. There is evidence to show that it will occur in hungry children or in those whose sucking time is insufficient. There is little need for treatment at this age, aside from making sure that the infant is reasonably well fed and is contented. Restraints do little good at this age and are definitely contraindicated at later ages. From 2 to 5 years, thumb and finger sucking are seen most commonly as more or less simple reactions to boredom, fatigue, illness, frustrations, deprivations and punishments. The habit is definitely used as a solace by the child. Treatment should be largely limited to ameliorating the more obvious sources of emotional dissatisfaction in the child. All semblance of a power contest between parent and child should be avoided. A direct attack on the symptom is most often of little use. In older children the symptom is most commonly seen as a part manifestation of a general emotional and social immaturity, which calls for detailed study and a broad program of personality and environmental readjustment. The pediatrician can do much to allay parental fears and to reassure the parents of younger children as to the harmlessness of the habit. The ill consequences of the habit which have been broadcast by the alarmists have, for the most part, no basis in fact. The deformity of the mouth is characteristic but will usually disappear spontaneously if the sucking stops before the eruption of the permanent teeth.

Obituaries

MARTHA WOLLSTEIN, M.D.

1868-1939

Dr. Martha Wollstein died in New York on September 30, 1939, after an illness of a few days' duration. In 1935 she retired from active work as pathologist of the Babies Hospital and assistant professor of pathology and of diseases of children in Columbia University, and since then she had been living part of the time in Grand Rapids, Mich., and part of the time in Flushing, N. Y.

After graduation from the Woman's Medical College of New York in 1889, Dr. Wollstein became the first resident physician of the newly organized Babies Hospital in 1890. Her interest in the morphology of disease led to her appointment as pathologist of the hospital in 1892, a post which she occupied for forty-three years. She held a teaching appointment on the staff of the Woman's Medical College in the nineties, was an associate of the Rockefeller Institute for Medical Research from 1906 to 1921 and later was connected with the department of pathology and the department of pediatrics of the College of Physicians and Surgeons after the affiliation of the Babies Hospital with Columbia University.

She was a well known pathologist, and at the time of her retirement she probably had had a more extensive experience in the morphology of disease in infants and children than any other American living. A bibliography of her writings, comprising sixty-eight items, contains thirty-six titles of studies devoted to descriptive and experimental pathology. She was especially interested in tuberculosis, dysentery, pyloric stenosis and leukemia and other diseases of the blood. In the field of bacteriology she was almost equally active, having carried out a number of investigations of the flora of the alimentary and the respiratory tract in relation to disease. Her experimental work on mumps was well known, and she contributed articles on this infection for two of the more widely used clinical compendia. Of a modest, retiring disposition, she avoided public demonstrations and speechmaking as far as possible; but with those who sought her help as man to man she eagerly shared her wide store of knowledge with open generosity.

She was a member of the New York Academy of Medicine, the New York Pathological Society, the American Association of Pathologists and Bacteriologists and the American Pediatric Society.

R. M.

Abstracts from Current Literature

Biochemistry; Bacteriology, and Pathology

CULTIVATION OF TRICHOMONAS FOETUS IN THE CHICK EMBRYO. P. M. NELSON, Proc. Soc. Exper. Biol. & Med. **39**:258 (Nov.) 1938.

Following the window technic of Goodpasture for the cultivation of virus in the living chick embryo, Nelson was able to grow Trichomonas foetus through fourteen generations by inoculating the parasite beneath the chorioallantoic membrane of the 11 or 12 day old chick embryo. HANSEN, Minneapolis.

ISOLATION OF LISTERELLA MONOCYTOGENES FROM INFECTIOUS MONONUCLEOSIS. C. A. PONS and L. A. JULIANELLE, Proc. Soc. Exper. Biol. & Med. **40**:360 (March) 1939.

A 16 year old white girl suffered from chills, fever, malaise and sore throat and had enlarged cervical and axillary lymph glands and a palpable spleen. Culture of blood taken on the ninth day of the disease revealed small gram-positive rod-shaped organisms resembling Listerella monocytogenes. The morphologic character of the blood and the agglutination of sheep's red blood cells (the heterophil test), with the clinical symptoms, justified the diagnosis of infectious mononucleosis. The organism isolated from the blood was fatal for rabbits, guinea pigs and mice, with lymphocytic-monocytic changes being induced in the blood. The culture was found to agglutinate in homologous antimonocytogenes serum. Various studies coincided in identifying the organism as L. monocytogenes. Instillation of the organism into the conjunctival sac of rabbit and the guinea pig produced purulent conjunctivitis. The patient recovered, and blood taken six weeks later showed no agglutination for the organism isolated or for sheep's red blood cells.

HANSEN, Minneapolis.

IDENTIFICATION OF LISTERELLA MONOCYTOGENES. L. A. JULIANELLE and C. A. PONS, Proc. Soc. Exper. Biol. & Med. **40**:362 (March) 1939.

A strain of Listerella monocytogenes isolated from the blood of a patient with infectious mononucleosis as well as seven strains from other animal sources have all been found to cause a typical conjunctival reaction, which is reproduced with equal readiness in the rabbit, guinea pig and rat. These organisms show the agglutination, the fermentative reactions and the stimulation of mononucleosis in susceptible animals characteristic of that bacterium. In producing the disease, young cultures are instilled into the conjunctival sac or swabbed on the everted lid. The rabbit is the most convenient animal to employ. A distinct conjunctivitis becomes visible in from one to five days, with acute and intense inflammation. The cornea becomes turbid in twenty-four to thirty-six hours and may appear roughened or pitted; blood vessels begin at the superior limbus and reach the pupillary margin. The acute reaction lasts five to ten days and subsides completely in one to three weeks. Histologic examination of the cornea shows infiltration by monocytes and invasion by small capillaries. The inflammation remains localized to the eye. Numerous other bacteria failed to produce such a change when instilled into the conjunctival sac. HANSEN, Minneapolis.

Metabolism; Infant Feeding; Milk and Other Foods

FURTHER STUDIES OF THE CALCIUM CONTENT OF THE BODIES OF RATS AS INFLUENCED BY THAT OF THE FOOD. C. S. LANFORD and H. C. SHERMAN, J. Biol. Chem. **126**:301 (Nov.) 1938.

Lanford and Sherman studied the effect of threefold and fourfold enrichments of the calcium content of an adequate diet on the calcium content of the bodies of rats at different ages. Marked differences in body calcium were shown in rats on the enriched as compared with those on an "adequate" diet. This difference was less marked as the animals grew older but persisted into adult life. The rate of calcification was more rapid on the higher calcium intake. The authors believe that this higher calcium content is advantageous throughout life.

LIMPER, Louisville, Ky.

WASTING OR MARASMUS. A. MONCRIEFF, Practitioner **142**:413 (April) 1939.

Failure to gain weight is a common complaint in infancy and childhood, and the wasting child is always a source of anxiety to its parents, the fear of tuberculosis being usually present in their minds, even if not voiced openly. Sometimes there is a finding that easily explains the wasting, such as an acute illness or an insufficient intake of food, but more usually the cause of the wasting is not obvious and the very use of the term "marasmus" in speaking of infants suggests profound ignorance of the etiologic factors. Strictly applied to a condition in which, despite an adequate intake of food and no obvious excessive loss by diarrhea and no evidence of infection, there is a complete failure of the weight to increase, the term is justified by long usage, but such a condition of defective utilization of food by the body tissues is rare. Only too often the infant whose condition is labeled "marasmus" is starving or is suffering from some infection.

The general causes of wasting include defective intake, digestion, absorption, transport and utilization and excessive expenditure, fever and toxemia.

GELSTON, San Francisco.

DIET IN HEALTH AND DISEASE: XXII. SPECIAL DIETS IN CHILDHOOD. W. W. PAYNE, Practitioner **142**:525 (April) 1939.

In constructing special diets it must be realized that there is interference with the free choice of both the nature and the amount of food taken. It is important, therefore, to be certain that the necessary amounts of salts and vitamins are given. To insure the correct amount of salts, an adequate amount of milk is given whenever possible. With regard to vitamins, A and D are best given in concentrated forms. There are many commercial preparations which are suitable. Vitamin B can be given as marmite, which has no food value. In salt-free diets, however, this product cannot be used, as it contains much salt. Vitamin C occurs in most diets in the daily fruit allowance.

Complete sample menus are given for fat-free diets, low residue diets, celiac disease and nephritis diets and high protein and ketogenic diets.

GELSTON, San Francisco.

ANTIBACTERIAL PROPERTIES OF COLOSTRUM. L. LUZZATTI, Riv. di clin. pediat. **37**:97 (Feb.) 1939.

It is possible to find bacteriolytic amboceptors against typhoid bacilli in colostrum. If complement is present, lysis takes place. But if complement is not present, colostrum acts as a good culture medium for Bacillus typhosus or Strepto-

coccus pyogenes. The immune bodies of colostrum act after their absorption into the blood of the offspring, rather than directly on the intestinal bacteria.

HIGGINS, Boston.

OCCURRENCE OF EOSINOPHILS IN SHORT PERIODS OF STARVATION. H. G. SOWKA, Folia haemat. **61**:106, 1938.

Sowka found in experiments on himself that starvation for twenty hours caused the percentage of eosinophils in his blood to rise on the average from 5 to 15. Alkaline ash and acid ash diets did not affect the percentage. Experiments on 3 friends did not show any change in the percentage of their eosinophils with starvation. Sowka concludes that because of mild vagotonia in himself starvation caused catabolism of body fat more quickly than in normal persons and that this accounted for the abnormality in the percentage of eosinophils.

LIMPER, Louisville, Ky.

CONGENITAL HYPOGLYCEMIA. W. EHRICH, Klin. Wchnschr. **13**:584 (April 21) 1934.

A diabetic mother aged 34 was delivered of her second child by cesarean section, owing to placenta praevia. Directly after delivery her blood sugar rose from 130 to 175 mg. per hundred cubic centimeters. The newborn infant had cramps at half hour intervals that were suggestive of hypoglycemic colic. The urine showed no sugar. Death occurred after twelve hours.

Autopsy showed a boy 50 cm. long, weighing 3,100 Gm. The lungs showed some atelectasis. The thymus and adrenals showed nothing remarkable. The head was not examined. Histologic sections of the pancreas showed enormous enlargement and increase in number of the islands of Langerhans.

SILVERMAN, Syracuse, N. Y.

CONSIDERATIONS IN INFANT NUTRITION. E. MAURER, München. med. Wchnschr. **85**:663 (May 6) 1938.

The success of the government of the Third Reich in increasing the birth rate has been the first step in preventing the national death of Germany. It is important that these newborn infants be saved and properly nourished. The infant mortality and the high incidence of rickets indicate that child welfare is not as good as it should be. Only 10 per cent of mothers nurse their infants for six months. About 10 per cent of mothers are unable for good reasons to nurse their infants. The other 80 per cent should be induced to nurse their infants as a duty. Maurer advises extensive propaganda for breast feeding and financial rewards to poor mothers. Infants should be nursed six times a day for two or three months to stimulate lactation. All mothers should be registered and weaning permitted only with a physician's consent. The people should be told that breast feeding is important not only for the infants but for the future generations. There is a lengthy discussion on the practical application of this idea.

Expensive proprietary milk preparations are used too frequently in place of cow's milk. Relatively few children will not thrive on cow's milk. Many mothers find that their children do not like to eat fresh vegetables. As a substitute they give them a proprietary combination of flour and vegetables which has a pleasant taste. Maurer questions the nutritional value of the products of this type which are extensively advertised.

BRAHDY, Mount Vernon, N. Y.

EFFECT OF THIRST AND SALT DEFICIENCY ON THE WATER DEPOTS OF THE ORGANISM. Ö. KERPEL-FRÓNIUS, Orvosi hetil. **82**:657 (July 2) 1938.

Kerpel-Frónius found in experiments on animals that the loss of water in thirsting affects both water depots (the extracellular and the intracellular) of the

organism equally, whereas when the loss of water is associated with salt deficiency—e. g., in cases of vomiting or diarrhea—circulation troubles occur much earlier, because in 90 per cent of these cases the loss of water is a loss of extracellular water.

GÖTTCHE, Budapest, Hungary.

SULFUR AND PROTEIN METABOLISM OF NATURALLY AND ARTIFICIALLY FED INFANTS. S. BLAZSÓ, Orvosi hetil. **82**:1188 (Dec. 3) 1938.

An examination of the sulfur metabolism of developing organisms showed that naturally fed infants retain almost the whole (90 per cent) of the sulfur-protein components of their food. Older children retain much less (15 per cent) and adults retain little (only 3 to 5 per cent). Examination of artificially fed infants showed that they retain only 50 to 60 per cent of the sulfur-protein components. This difference in retention leads to the supposition that artificially fed infants are unable to use up the sulfur-protein content to the degree that the naturally fed infants can use up the sulfur-protein components of mother's milk.

GÖTTCHE, Budapest, Hungary.

Vitamins; Avitaminoses

CAN RICKETS BE ELIMINATED FROM THE LARGE CITIES? J. LEVY and H. B. SILVER, Arch. Pediat. **56**:96 (Feb.) 1939.

After observing 1 case of rickets in 100 unselected cases the authors conclude that the disease may be eradicated from large cities.

ORR, Buffalo.

VITAMIN B FRACTIONS AND INSULIN TOLERANCE IN THE ALBINO RAT. A. R. McINTYRE and J. C. BURKE, J. Pharmacol. & Exper. Therap. **65**:36 (Jan.) 1939.

Undernourished infants have been given insulin in an attempt to increase the utilization of carbohydrate. The early seemingly favorable reports have not been confirmed. The authors investigated the effects of insulin on growing rats under many conditions, with conclusions as follows:

1. Daily administration of 8 units of insulin per kilogram of body weight does not alter the rate of weight increase in albino rats 4 to 5 months of age when they are fed a normal diet ad libitum.

2. Albino rats of this age will tolerate at least 200 units of insulin per day in single doses for five days without exhibiting serious depression or convulsions if given a normal diet ad libitum.

3. Rats totally or gradually starved but allowed water ad libitum will tolerate 20 units of insulin per kilogram per day for five days unless their loss in weight by the first day of injection has reached 25 per cent or more of their initial weight.

4. Rats deprived of all the vitamin B complex or of the heat stable factors, with or without flavin, are hypersensitive to insulin. This increased sensitivity is not explainable on the basis of inanition and loss in body weight alone.

5. The presence of thiamin in an otherwise vitamin B–deficient diet increases the sensitivity of the rat to insulin.

PILCHER, Cleveland.

VITAMIN C NUTRITION IN ARTIFICIAL FEVER. J. ZOOK and H. SHARPLESS, Proc. Soc. Exper. Biol. & Med. **39**:233 (Nov.) 1938.

Guinea pigs on a scorbutic diet when subjected to artificial fever were depleted of their vitamin C stores faster than control animals. The losses in the adrenals were very marked. In 6 human subjects the urinary excretion of vitamin C was decreased following artificial fever, while the blood values were unchanged. These experiments indicate that the physiologic need for vitamin C is increased during fever.

HANSEN, Minneapolis.

EFFECT OF VITAMIN B_1 AND VITAMIN B_2 COMPLEX ON THE LOSS OF WEIGHT PRODUCED IN RATS BY EXPERIMENTAL HYPERTHYROIDISM. V. A. DRILL, Proc. Soc. Exper. Biol. & Med. **39**:313 (Nov.) 1938.

Female adult rats (weight, 250 Gm.) were given 100 mg. of thyroid gland daily in addition to their normal diet. These animals lost weight rapidly, 23 Gm. per rat in seventeen days. Twelve test rats were given 500 Gm. of vitamin B_1 daily injections, and they stopped losing weight, while the controls, receiving no vitamin B_1 continued to lose weight. On the thirtieth day, a brewers' yeast concentrate was added to the diet in the proportion of 5 per cent, and an immediate gain in weight was noted. Male rats in the same experiment did not gain weight when vitamin B_1 and a yeast concentrate were added to the diet of animals receiving thyroid gland extract daily; thus a sex difference is noted. However, in a third experiment, when the thyroid extract was reduced to 50 mg. daily, a weight increase occurred when both vitamin B_1 and the vitamin B_2 complex were administered.

HANSEN, Minneapolis.

INACTIVATION OF VITAMIN K BY LIGHT. D. W. MACCORQUODALE, S. B. BINKLEY, R. W. MCKEE, S. A. THAYER and E. A. DOISY, Proc. Soc. Exper. Biol. & Med. **40**:482 (March) 1939.

Two preparations of vitamin K, a noncrystalline product from alfalfa and a crystalline product from putrefied fish meal, were found to undergo great destruction when exposed to sunlight, white light and light from a 300 watt daylight bulb. A preparation kept in the dark contained ten times the potency of a control one exposed to light.

HANSEN, Minneapolis.

STUDIES IN THE URINARY EXCRETION OF VITAMIN C IN CERTAIN DISEASES OF CHILDREN. S. WILLIAMS and M. GREEN, M. J. Australia **1**:145 (Jan. 28) 1939.

The daily excretion of vitamin C in the urine of normal children was found to be quite constant for each child but to vary within a range of 300 per cent in different ones. The usual test for deficiency was used, i. e., the measure of urinary excretion after the administration of a test dose of ascorbic acid.

Vitamin C deficiencies have been shown to exist in many diseases, but these authors feel that "commercial enterprise is ahead of experimental justification for the general use of synthetic products." The results of tests in various diseases show no deficiency of vitamin C in stomatitis but considerable deficiency in hemorrhagic purpura and in scurvy. Curiously enough, a patient with scurvy failed to respond to ascorbic acid but did respond to orange juice. The authors have found the intradermal test for vitamin deficiency, i. e., decolorization of dichlorophenolindophenol, unreliable, as is also the capillary resistance test, because so many factors enter into the question of capillary resistance.

TENNEY, Madison, Wis.

VERIFICATION OF TESTS OF HYPOVITAMINOSIS C IN CHILDREN OF SCHOOL AGE. NEIMANN and DEDUN, Rev. franç. de pediat. **14**:253, 1938.

The authors review the various tests for vitamin C deficiency and choose from these the two most simple for their study of 207 children. The first is that of Goethlin, in which a count is made of the number of petechiae developing as a result of partial obstruction of the venous return in the arm. This obstruction is obtained by the use of a blood pressure cuff maintained at a pressure of 50 mm. of mercury for fifteen minutes. A count of over 5 is said to indicate vitamin C deficiency. The second test is that of Tillmanov in which the vitamin C content of the urine is measured. A value under 1 mg. per hundred cubic centimeters is said to indicate deficiency. Only 9 children showed such a value, whereas 40

showed over 5 petechiae. The authors conclude that neither test is absolute but that both may serve in indicating children of whom further study is desirable.

LESLIE, Evanston, Ill.

VITAMIN B_1 AND GLYCEMIA. A. LIOTTA, Riv. di clin. pediat. **37**:62 (Jan.) 1939.

The blood sugar of pigeons and rabbits during fasting was unchanged in amount after the injection of 100 and of 1,000 international units of vitamin B_1.

Studies of the blood sugar curves in pigeons and rabbits following the ingestion of sugar and as affected by vitamin B_1 and insulin indicate that vitamin B_1 usually has an action antagonistic to insulin when rabbits are fasting and synergistic to insulin when sugar is given.

The action of vitamin B_1 on sugar metabolism is probably through the sympathetic nervous system.

HIGGINS, Boston.

VITAMIN C CONTENT OF CANNED VEGETABLES. H. BISCHOFF and L. GRASEDYCK-RENNER, Monatschr. f. Kinderh. **78**:45 (March) 1939.

The writers examined a variety of canned vegetables and found that the specimens examined contained fairly adequate quantities of vitamin C.

GERSTLEY, Chicago.

SIGNIFICANCE OF THE RACHITIC BONE CHANGES IN EARLY CHILDHOOD FOR LATER LIFE. M. LANGE, München. med. Wchnschr. **84**:2022 (Dec. 17) 1937.

Since good rachitic prophylactic measures have been in use for twenty years, one would expect an absence of late effects of infantile rickets. This expectation is only partly met. One no longer sees the most marked rachitic bone changes, but there are too many of the moderate type. Lange describes the changes which he has observed: those involving the extremities, spine, pelvis and thorax.

BRAHDY, Mount Vernon, N. Y.

THE PROBLEM OF CURING RICKETS. E. THOMAS, München. med. Wchnschr. **85**: 662 (May 6) 1938.

The rickets of infancy may heal spontaneously or remain latent with or without specific antirachitic therapy. Infants treated for rickets may present bony deformities due to rickets later in childhood. Thomas emphasizes the importance of medical supervision during childhood.

BRAHDY, Mount Vernon, N. Y.

THERAPY AND PROPHYLAXIS OF RICKETS WITH A SINGLE LARGE DOSE OF VITAMIN D_2 AND D_3. H. BROCKMANN, Ztschr. f. Kinderh. **60**:359 (Dec. 19) 1938.

Brockmann was able to substantiate the results of other authors regarding the rapidly curative effect on rickets of a single large dose of vitamin D. Prophylactically, a single large dose protects for at least two and one-half months. When given in large doses, vitamin D_3 and D_2 are of equal effectiveness. Brockmann recommends giving large doses in mid-October and in the latter part of January as prophylactic treatment.

SIEMSEN, Chicago.

Prematurity and Congenital Deformities

FACTORS INFLUENCING THE MORTALITY RATE OF PREMATURE INFANTS. T. M. LAMB, Brooklyn Hosp. J. **1**:69 (April) 1939.

This report concerns 120 premature babies born at the Brooklyn Hospital since January 1936. Ten of these died, 8 within the first twenty-four hours, leaving only 2 deaths among those who survived the first day. One of these had a con-

genital lesion of the heart and the other a cerebral hemorrhage. Of the 8 deaths occurring within the first twenty-four hours, 2 occurred in babies delivered by cesarean section from mothers who had received morphine and were under general anesthesia. It is thought that the delivery of choice for a premature baby is that with low forceps control and episiotomy, since it shortens the period in which the head is pressing on the perineum.

The excellence of the results obtained in this series of cases is thought to depend partly on the maintenance of a relative humidity of 55 per cent in the air surrounding the infants, partly on maintaining the infants at a temperature varying between 98.6 to 100.6 F., partly on handling the infants as little as possible (no infant weighing under 5 pounds [2,268 Gm.] is examined by the physician unless there is a very definite indication for it) and partly on expert gavage feeding, in which the amount of food given depends on what the infant can take care of rather than on any theory as to the amount required. An isolation unit was maintained for babies with evidence of infection.

LESLIE, Evanston, Ill.

ARACHNODACTYLIA. H. J. WYCKOFF, Northwest Med. **38**:134 (April) 1939.

Arachnodactylia is described as observed in 3 children. Each showed spinal changes, disturbance in extremities, visual disturbance and abnormal cardiac conditions. This is undoubtedly a congenital abnormality and due probably to some disturbance occurring at the time of development of the three primary germ layers.

Little in the way of treatment is recommended except the correction of deformities in patients whose disability is sufficient to warrant it.

ROBERTSON, Omaha, Neb.

INFANT MORTALITY IN BATAVIA FOR THE YEARS 1935-1936. J. H. DE HAAS, Indian J. Pediat. **6**:12 (Jan.) 1939.

In 1935-1936 the city of Batavia had among its native, Chinese and European population infant mortality rates of + 30 per cent, 10 per cent and 5.6 per cent, respectively. Among the native population the infant mortality rate of 300 per thousand live born infants is as high as, if not higher than, that in other cities in the tropics.

GONCE, Madison, Wis.

A CASE OF TERATOMA OF THE NECK OF A FOETUS WITH ACUTE HYDRAMNIOS. J. ST. GEORGE WILSON, J. Obst. & Gynaec. Brit. Emp. **46**:44 (April) 1939.

A stillborn fetus weighing 2½ pounds (1,134 Gm.), with a tumor on the anterior surface of the neck measuring 4 by 4½ inches (10 by 11.5 cm.), is described. The tumor was attached to the base of the tongue and front of the larynx. It was composed of embryonic cartilage with some ossification, neuroblastic tissue and tissue suggestive of the large intestine. The diagnosis was teratoma of the neck.

The tumor was not associated with the thyroid gland, and the author believes that in many cases in which a tumor is described as teratoma of the thyroid it actually has no relation to the thyroid.

POTTER, Chicago.

EVIDENCE OF FOETAL RESPIRATION IN UTERO. T. H. BELT, J. Obst. & Gynaec. Brit. Emp. **46**:316 (April) 1939.

A maternal hemorrhage and an infarct discovered in the placenta at the delivery of a full term infant are believed to have caused transitory anoxemia in the infant at the time the hemorrhage occurred. The infant was stillborn. At autopsy the lungs showed subpleural nodules composed of lymphoid tissue and cells from the amniotic fluid. Belt concludes that the infant aspirated amniotic fluid during the period of anoxemia and that the nodules were a response to the amniotic cells.

POTTER, Chicago.

SUMMER CAMPS FOR CHILDREN. I. NEMOIANU, Med. inf., Roumania **1**:104 (Feb.) 1939.

According to the model for camps for children organized in Italy, one can distinguish three types: permanent, temporary and day camps.

Permanent camps correspond in reality to hospitals. The duration of the sojourn varies from three to four months to one to two years. Temporary camps receive in general lymphatic and anemic children or those who are carriers of tracheobronchial adenopathies. The minimum duration of sojourn is forty days. The day camps with a prophylactic purpose receive children living in hygienic surroundings which are inadequate and children with lowered nutrition. The day camps should be situated where there is a forest, a lake, gardens, a park or other such natural feature. The choice of the location for the establishment of the camp depends on the climate. Small camps are to be preferred, taking 25 to 30 children and not necessitating large establishments. For day camps, wood barracks are entirely sufficient. The physician of the camp ought to indicate heliotherapy, gymnastics, sports and medical treatment. In Rumania, where day camps ought to be the centers of hygienic and moral education, there should be more encouragement of the founding of such camps. FROM AUTHOR'S SUMMARY.

ARACHNODACTYLIA IN TWO SIBLINGS. E. SCHMIEDING, Monatschr. f. Kinderh. **78**:56 (March) 1939.

Schmieding reports archnodactylia in brother and sister, aged 7 and 5, respectively, with photographs. GERSTLEY, Chicago.

Newborn

PANEL DISCUSSION ON CYANOSIS OF THE NEWBORN. C. F. MCKHANN, New England J. Med. **219**:899 (Dec. 8) 1938.

About 5 Gm. of reduced hemoglobin per hundred cubic centimeters of capillary blood is necessary to produce a bluish appearance of the skin and mucous membranes. In other words, in a person with a normal level of hemoglobin about one third of the blood must be unsaturated with oxygen (reduced) before cyanosis appears.

It is to be noted that whereas maternal arterial blood is probably 95 per cent saturated with oxygen, fetal arterial blood is to a considerable degree unsaturated, so that at the time of birth the fetus may actually have physiologic cyanosis. However, the oxygen supply to the tissues is not inadequate because of the large amount of hemoglobin in the fetal circulation and the increased total capacity.

GENGENBACH, Denver.

PANEL DISCUSSION ON CYANOSIS OF THE NEWBORN: CYANOSIS FOLLOWING INTRACRANIAL INJURIES. S. H. CLIFFORD, New England J. Med. **219**:900 (Dec. 8) 1938.

Intracranial injury may produce cyanosis when there is direct or indirect involvement of the respiratory center. The respiratory center may be affected by the pressure of gross hemorrhage or of gross cerebral edema.

The two major causes of cerebral injury are trauma and asphyxia, either or both contributing to the injury. From the practical point of view, both trauma and asphyxia are usually present with intracranial hemorrhage. In reviewing 100 autopsies on infants with intracranial hemorrhage no case was found in which the lesions could be explained on the basis of trauma alone.

The prognosis is dependent on the location and degree of the injury. A diffuse hemorrhage in the subarachnoid or subdural spaces carries an excellent prognosis, provided the injury is limited to these regions. A hemorrhage of less degree into the region of the medulla, however, is nearly always fatal. During 1936 at the

Boston Lying-in Hospital there was but 1 death from the traumatic type of intracranial hemorrhage in nearly 4,000 births. The problem of reducing the intracranial injuries due to intrauterine asphyxia, however, is still unsolved.

GENGENBACH, Denver.

PANEL DISCUSSION ON CYANOSIS OF THE NEWBORN: CIRCULATORY CAUSES OF CYANOSIS. H. GREEN, New England J. Med. **219**:901 (Dec. 8) 1938.

Cyanosis of the newborn caused by disturbance of the peripheral circulation may be due to trauma, infection or congenital malformation.

A complete examination of the blood should be done. This will rule out erythroblastosis foetalis. Red cell counts and hemoglobin estimations are most important, as they are particularly high in congenital heart disease. Cardiac enlargement shown by roentgen rays in a newborn baby ostensibly means congenital heart disease. The electrocardiograph has been of some value in determining congenital heart disease but not particularly in the newborn. If a newborn baby has cyanosis, dyspnea, a high red cell count, trouble in feeding, multiple congenital deformities and roentgen evidence of cardiac enlargement with or without a murmur, it should be assumed to have a congenital malformation of the heart.

GENGENBACH, Denver.

PANEL DISCUSSION ON CYANOSIS OF THE NEWBORN: PULMONARY CAUSES OF CYANOSIS. J. M. BATY, New England J. Med. **219**:903 (Dec. 8) 1938.

Because of various factors interfering with respiration, physiologic atelectasis may persist to a pathologic extent and be of distinct importance in the production of cyanosis. Wilson and Farber considered the causes of atelectasis to be as follows: (1) cohesion of the moist surfaces of the air passages in collapsed and airless lungs; (2) imperfect development or injury of the respiratory center; (3) poor development of the thorax, seen especially in premature infants, and (4) bronchial obstruction due to aspiration of the contents of the amniotic sac, mucus or blood.

GENGENBACH, Denver.

PANEL DISCUSSION ON CYANOSIS OF THE NEWBORN: PATHOLOGICAL ASPECTS OF CYANOSIS. S. FARBER, New England J. Med. **219**:904 (Dec. 8) 1938.

Cerebral hemorrhage, edema and congestion following obstetric trauma or intrauterine asphyxia are the lesions most commonly found as the cerebral causes of cyanosis. In general, massive cerebral hemorrhage secondary to demonstrable gross tears of the large cerebral veins may be ascribed to obstetric trauma. Smaller areas of hemorrhage, usually multiple in nature, are most often associated with intrauterine asphyxia, to which a traumatic factor may be added. Pneumonia in the newborn is rarely the result of a diaplacental infection. It occurs most often as a consequence of aspiration of infected amniotic sac contents or of gross exposure of the newborn to pathogenic organisms. The finding of large amounts of amniotic sac contents in the lungs constitutes the most reliable evidence that intrauterine asphyxia has occurred. Pulmonary congestion, edema and hemorrhage may occur following parenteral administration of blood and other fluids in amounts too large for the circulatory system of the newborn.

The circulatory system is responsible for cyanosis in the newborn when important congenital defects are present. Cyanosis does not usually occur until more than 30 per cent of the venous blood has been shunted into the arterial circulation. Extrinsic obstruction to the entrance of air to the lungs may be caused by a vascular circle around the trachea. The thymus has not been found a cause of cyanosis.

There appears to be sufficient evidence for one to speak of "immaturity of the respiratory center" in very small premature babies. GENGENBACH, Denver.

PANEL DISCUSSION ON CYANOSIS OF THE NEWBORN: OBSTETRIC ASPECTS OF CYANOSIS. H. M. TEEL, New England J. Med. **219**:906 (Dec. 8) 1938.

The chief obstetric causes of disturbances in the newborn infant of which abnormal cyanosis is a symptom may be presented logically under the general headings intrauterine asphyxia, birth trauma and drugs.

Intrauterine asphyxia is the immediate cause of practically all stillbirths, including those of macerated fetuses. Common causes of such asphyxia are: anesthesia of the mother for a considerable interval before delivery; premature separation of the placenta, infarcts of the placenta and tetanic contraction of the uterus, as occasionally seen after the use of posterior pituitary in labor; prolapse of the cord, due to looping of the cord about the neck. The most important diagnostic signs of distress of the fetus in utero are acceleration, slowing or gross irregularity of the fetal heart beat, in the order named. For this reason it is important to listen to the fetal heart at increasingly frequent intervals as labor progresses.

Trauma incident to labor and operative delivery is another frequent cause of abnormal cyanosis of the newborn. The commonest lesions are intracranial hemorrhages due to stresses applied to the fetal head.

The use of sedative, analgesic and anesthetic drugs during labor is a third obstetric cause of abnormal cyanosis in the newborn. It has been shown that the respiratory mechanism of the fetus in utero is highly susceptible to such drugs given to the mother. The giving of opiates within four hours before delivery is often followed by marked and sometimes alarming asphyxia neonatorum in mature babies.

GENGENBACH, Denver.

TWO CASES OF GASTROINTESTINAL HEMORRHAGE OF THE NEWBORN. B. TASSOVATZ and S. TASSOVATZ, Bull. Soc. d'obst. et de gynéc. **27**:641 (Oct.) 1938.

This report is from Belgrade. The methods of treating gastrointestinal hemorrhage of the newborn are reviewed. The authors mention the use of horse serum, gelatin, epinephrine, human serum or whole blood, transfusion and administration of several proprietary preparations. Of these transfusion is the most important, but with the newborn the technical difficulties of transfusion are so great that it is reserved only for the most severely affected. Two cases of hemorrhagic disease of the newborn in which the gastrointestinal tract was involved are reported. The infants were given adult human blood, calcium, gelatin and several proprietary preparations, with recovery of both.

GLASER, Rochester, N. Y.

ENORMOUS HEMATOMA OF THE SCALP RESULTING IN DEATH. EPARVIER and NOTTER, Bull. Soc. d'obst. et de gynéc. **27**:768 (Dec.) 1938.

The patient was a boy delivered normally from a left occipital-anterior position in a 28 year old primipara. The birth weight was 3,850 Gm. At birth a hematoma of the scalp, the size of half an orange, was noted at the base of the occiput. Otherwise the infant appeared normal. During the night the hematoma spread enormously, dissecting the scalp completely from the cranium and covering the head like an enormous helmet. The infant became very pale and died apparently from loss of blood into the hematoma. At autopsy no abnormality was found except the hematoma. The authors do not suggest a possible cause of the hemorrhage or what vessels might have been involved. In the discussion of this presentation 4 similar cases are mentioned.

GLASER, Rochester, N. Y.

UREA CONTENT OF THE BRAIN OF THE NEWBORN. C. BRAENDL, Klin. Wchnschr. **13**:622 (April 28) 1934.

No relationship was found between the urea content of the brain and the weight of the body. The greatest amounts were found in those cases in which uric acid infarcts were found in the kidneys.

SILVERMAN, Syracuse, N. Y.

PRECAUTION IN THE HANDLING OF THE NEWBORN. H. HOFWEBER, München. med. Wchnschr. **85**:789 (May 27) 1938.

Hofweber discusses certain procedures in the care of a newborn infant with cerebral hemorrhage or of one who is likely to have a cerebral hemorrhage. The measures for resuscitation described by Schultze and by Prochownick are condemned. Any procedure in which the feet are elevated above the head is harmful. Resuscitation can be helped by placing the infant in a warm bath, thereby drawing blood from the brain to the periphery. Further stimulation can be obtained by putting the infant in cool water, which will draw the blood to the surface of the body. Indicated medication can be injected into the buttocks or scalp during delivery. The infant can be kept in the warm bath for a prolonged period before it is placed in a warmed bed with the head-end elevated.

BRAHDY, Mount Vernon, N. Y.

CLINICAL AND HISTOLOGIC INVESTIGATIONS OF THE SO-CALLED WITCH'S MILK LACTATION WITH PARTICULAR ATTENTION TO ITS RELATION TO THE BIRTH WEIGHT OF THE INFANT. P. FORSSELL, Acta pædiat. (supp. 1) **23**:1, 1938.

This is a monograph of 106 pages, amply documented and well illustrated. The conclusions are based on clinical observation of 1,063 infants and histologic examination of the breasts of 92 infants that died of various causes during the first year of life. The study was made at the Ylppö clinic in Helsingfors.

Lactation occurs in all newly born infants except those whose birth weight is under 1,500 to 1,600 Gm.; in this group the phenomenon is rare. The occurrence of lactation is no more common in infants weighing more than 4,000 Gm. at birth than in those whose birth weights are closer to the average. Secretion usually begins on the third or fourth day of life. In about 10 per cent of the cases it appears on the first day and in a small proportion as late as the eighth day. Rarely it may not be observed until during the second or third week of life. It commences somewhat earlier in the larger infants than in the smaller ones; this correlation is related to the degree of development of the mammary apparatus. The time of appearance of the "witch's milk" is apparently not influenced by the state of health of the mother, by the length of labor or by the physical condition of the infant at birth. The duration of lactation was found to be considerably longer than has been reported by previous observers: The majority of the infants observed continued to secrete "witch's milk" for two months; between the ages 3 and 4 months it was still present in 75 per cent of the infants that were followed; between 8 and 9 months 24 per cent of the infants still showed a small amount of secretion. No difference as to sex was found in relation either to the time of appearance or to the duration of lactation. No correlation could be demonstrated between maternal and infantile lactation. In contrast with the time of onset of lactation, the duration of lactation was not found to be related to birth weight.

MCCUNE, New York.

CONGENITAL ANOMALY OF THE VALVE OF BAUHIN AND OF THE APPENDIX AS A CAUSE OF PERITONITIS IN THE NEWLY BORN. P. I. TUOVINEN, Acta pædiat. **23**:234, 1938.

Tuovinen reports briefly the case of a newly born infant who exhibited evidence of ileus, suspected to be due to peritonitis. Enterostomy gave temporary relief but failed to effect a cure, and the patient died on the eighth day of life. Autopsy revealed congenital atresia of the common bile duct and almost complete stenosis of the ileocecal valve; the appendix was bound down by mesenteric adhesions, and regional peritonitis was present.

MCCUNE, New York.

A CASE OF CONGENITAL ICHTHYOSIS. O. LUNDQUIST, Acta pædiat. **23**:252, 1938.

Although ichthyosis is not a particularly uncommon disease of the skin, the congenital form is somewhat rare. In this form the dermal alterations commence

as early as the fourth or the fifth month of intrauterine life and consist in marked thickening of the epidermis with resulting fissuring thereof and deformities of the eyelids, ears, nose and lips. The thickening affects principally the stratum corneum and occasionally the stratum germinativum; the stratum granulare and the stratum lucidum are apt to be underdeveloped. Severe forms may lead to premature parturition and death; the milder forms do not endanger life, but the skin never becomes entirely normal. The form which develops later, termed ichthyosis vulgare, is distinguished from the congenital form principally on the basis of time of onset and the severity. The etiologic factors in both forms remain completely obscure.

In the case that is reported the infant presented the characteristic cutaneous alterations of ichthyosis at birth; the distortion of the face was particularly conspicuous. The thickened skin had a parchment-like consistency. Evidence of syphilis was lacking. Erythrodermia ichthyosiforme congenita (Brock) was readily excluded on the basis of clinical features. Painstaking measures were carried out to prevent infection of the skin, and the infant was fed on breast milk. Within a few days the child's skin began to desquamate in great flakes; at three weeks the thick cuirass was replaced by a relatively normal skin which was only moderately ichthyotic. A brief bibliography is supplied.

McCune, New York.

Acute Contagious Diseases

Scarlet Fever Survey. S. Peacock, J. A. Bigler and M. Werner, Illinois M. J. **75**:46 (Jan.) 1939.

During 1934 and 1935 the Dick test was made on all children admitted to the Children's Memorial Hospital and on the whole hospital personnel, a cultural study was made of nasal and pharyngeal mucus for hemolytic streptococci, and all cases of scarlet fever occurring in the city of Chicago were reviewed.

Although the statistical observations are too detailed for abstracting, certain general observations of the authors are worthy of note. Too much reliance cannot be placed on a negative or a positive culture on any one day when one is considering a carrier of the hemolytic streptococcus. Mucus from infants is less likely to yield positive cultures than that from children. There is a gradual increase in the incidence of positive cultures up to the fourth year of age, after which the incidence remains about the same. The size of the family has no influence on the presence or absence of positive cultures, but there is a definite gradual decrease in the number of persons with positive reactions to the Dick test with increase in family size. Although it seems that the presence of tonsils and adenoids in children has a greater influence on the presence of hemolytic streptococci in their noses and throats than the presence of infection of the throat, this influence is not great enough to warrant advising tonsillectomy at an early age to prevent scarlet fever. The nose seems to be of relatively little importance when a carrier of hemolytic streptococci is being considered. There was a gradual decrease in the number of persons reacting positively to the Dick test from a high of 71 per cent in the 1 year old group to 28 per cent in the 12 year old group. There is a fairly high incidence of failure of immunity after the disease as measured by the Dick test. The information received from the interpretation of the Dick test is not always reliable. The monthly incidence of cultures positive for hemolytic streptococci follows quite closely the monthly incidence of the disease. The age curve of positive cultures also follows to some extent the age incidence of scarlet fever. The age incidence of positive reactions to the Dick test, however, in no way follows the age incidence of scarlet fever. In from 17.3 to 20 per cent of the reported cases of scarlet fever the infection could be considered as secondary, arising in a home following exposure to scarlet fever occurring primarily in some other member of

the family. More of the secondary cases in homes arose from contact with patients who had been quarantined in hospitals then from contact with those quarantined at home.

BARBOUR, Peoria, Ill.

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES. Pub. Health Rep. **54**:593 (April 14) 1939.

For the period of Feb. 26 to March 25, 1939, there were seven times as many cases of influenza (63,927) as in 1938. The greatest excess was in the South Atlantic, North Central and Mountain regions. The incidence of typhoid fever was 20 per cent and that of smallpox 30 per cent over the five year average. The number of cases of diphtheria, measles, poliomyelitis and meningitis were below median. The average mortality from all causes was 13 per thousand. This is slightly higher than the average.

SANFORD, Chicago.

A CASE OF RELAPSE IN EXPERIMENTAL POLIOMYELITIS. W. K. HEASLIP, Australian J. Exper. Biol. & M. Sc. **16**:285 (Dec.) 1938.

In a case of experimental poliomyelitis a relapse occurred three weeks after apparent recovery.

GONCE, Madison, Wis.

VITAMIN C NUTRITION AND SUSCEPTIBILITY TO DIPHTHERIA AS POSSIBLE FACTORS IN THE EPIDEMIOLOGY OF POLIOMYELITIS. W. K. HEASLIP, Australian J. Exper. Biol. & M. Sc. **16**:287 (Dec.) 1938.

On the basis of a study of the Schick reactions in 238 children with poliomyelitis and 207 nonimmunized controls, Heaslip offers evidence to show that there is a greater susceptibility to poliomyelitis in persons giving positive Schick reactions than in those who give negative reactions. Evidence is also presented to show that the urinary excretion of vitamin C is lowered in infectious states in general and that in the case of poliomyelitis the reduction in vitamin C is related to the susceptibility of the patient and to the severity of the lesions.

GONCE, Madison, Wis.

THE CASE FOR INDIVIDUAL ISOLATION: ITS USE IN INFECTIOUS DISEASES AND IN THE CARE OF INFANTS. H. J. SINN, M. J. Australia **1**:4 (Jan. 7) 1939.

Sinn discusses current methods of isolation under the headings "bed isolation," "disease isolation" and "individual isolation" ("cubicle system" and "separate room system"). Individual isolation is regarded to be the best method of treatment for infectious diseases, especially streptococcic infections, and for the nursing of young infants.

GONCE, Madison, Wis.

DISTURBANCE OF GAIT, PROBABLY FOLLOWING SCARLATINAL ENCEPHALITIS. S. Z. KULIEOWSKI, Rev. neurol. **70**:384 (Oct.) 1938.

A girl of 9 years contracted severe scarlet fever, in the course of which aphasia developed. Progressive difficulty in walking appeared. Three attacks of convulsions ensued. She presented hemiparesis on the right, holding the right leg in equinovarus with induction of the foot and outward rotation of the leg. The spinal fluid was normal. Encephalograms showed a moderate amount of cortical atrophy, most marked in the right motor areas.

DAFFINEE, Boston.

EMBOLUS OF THE FEMORAL ARTERY FOLLOWING CHICKENPOX. A. NACCARI, Méd. inf. **10**:8 (Jan.) 1939.

An unusual complication occurred in a 15 month old girl six days after the appearance of chickenpox, when gangrene of the left leg appeared about two thirds of the way down the thigh; amputation was necessary. The cause of the gangrene was probably an embolus in the lower part of the femoral artery. The embolus

perhaps originated from the neighborhood of an infected pock. A primary pathologic condition of the femoral artery or abnormal chemical composition of the blood may have been contributing factors.

HIGGINS, Boston.

CORTICAL FORM OF HEINE-MEDIN'S DISEASE. P. VON KISS and I. FENYES, Jahrb. f. Kinderh. **147**:97 (July) 1936.

Points regarding the controversy as to whether there is actually a cortical form of Heine-Medin disease are given. The authors feel that there is and give 7 detailed case reports in support of their contention.

LANDAU, Hannibal, Mo.

DIPHTHERIA IN NUREMBERG IN RECENT YEARS. H. J. SPÖRL, Jahrb. f. Kinderh. **147**:129 (Aug.) 1936.

There was no familial susceptibility to malignant diphtheria in the series of patients studied. In 90.7 per cent of the patients with severe pharyngeal diphtheria circulatory disturbances were noted consisting of extrasystoles, heart block and total arrhythmia. Vasomotor disturbances were found frequently. Renal changes characteristic of nephrosis were noted in several patients.

The desirability of giving specific therapy on definite clinical evidence of diphtheria despite negative laboratory findings is stressed. Early administration of serum is advised.

LANDAU, Hannibal, Mo.

VALUE OF ELECTROCARDIOGRAPHY IN DIPHTHERIA. C. KEPPEL, Jahrb. f. Kinderh. **147**:170 (Aug.) 1936.

The value of following patients with diphtheria by means of the electrocardiograph is emphasized. Case reports in illustration of toxic myocarditis, atrioventricular block (both complete and partial), sinus block and other conditions are given, with electrocardiograms.

The value of these tracings both in prognosis and in therapeutics is stressed. Keppel feels that the optimal time for allowing the child to return to normal activity can be determined more accurately by what the electrocardiogram shows.

DANDAU, Hannibal, Mo.

LOCAL AND PERIPHERAL LEUKOCYTE COUNTS IN CERTAIN INFECTIOUS DISEASES. K. SCHULTZE and K. STEUER, Klin. Wchnschr. **13**:407 (March 17) 1934.

From 25 patients (12 with scarlet fever, 8 with measles and 5 with typhoid) blood was taken for leukocyte and differential counts. The blood was obtained from the lobe of the ear and also from skin lesions (rose spots, papules and the like). No difference was found in the white cell count, but in the majority of instances a slight relative and absolute lymphocytosis was found in the "local" as compared with the peripheral blood.

SILVERMAN, Syracuse, N. Y.

WHEN SHALL DIGITALIS BE GIVEN IN DIPHTHERIA? P. VON KISS, Klin. Wchnschr. **13**:411 (March 17) 1934.

While no digitalis should be used in the treatment of patients with diphtheria without electrocardiographic control, the author recommends, on the basis of his experience with 20 such patients, the administration of small doses at the first electrocardiographic evidence of myocardial change.

SILVERMAN, Syracuse, N. Y.

DIPHTHERIA IN LÜBECK FROM 1910 TO 1937. W. GOETERS, Monatschr. f. Kinderh. **77**:38 (Jan.) 1939.

During the twenty-seven years 1910 to 1937 there were three epidemic peaks —1917-1918, 1928-1929 and 1935-1937. The severest mortality occurred in 1927 with a great increase in incidence of the toxic form of diphtheria; 62.5 per cent of

Goeters' 80 patients died. The majority of the toxic patients were aged between 6 and 9 years. Presumably the virulence of the epidemic was due to the virulence of the germ. After 1937 the incidence of the toxic form of the disease diminished, while laryngeal diphtheria increased.

GERSTLEY, Chicago.

DIPHTHERIA IN LÜBECK: 1937-1938. W. REINHARDT, Monatschr. f. Kinderh. **77**: 53 (Jan.) 1939.

After 1937 toxic diphtheria diminished rapidly in Lübeck. Of 445 patients, only 6 died.

For diagnosis, throat swabs were found more efficient than cultures.

No explanation is given for the sudden diminution in incidence of the toxic form of the disease other than the hypothesis of a sudden diminution in the virulence of the organism.

GERSTLEY, Chicago.

RELATION BETWEEN SUN SPOTS AND DIPHTHERIA. S. BELAK, Monatschr. f. Kinderh. **77**:190 (Feb.) 1939.

Some writers have attempted to establish a relation between sunspots and epidemics of diphtheria. Belak is unable to do this.

GERSTLEY, Chicago.

EXTENSION OF THE DISEASE PROCESS IN HEINE-MEDIN'S DISEASE. G. PETERS, München. med. Wchnschr. **85**:1073 (July 15) 1938.

Müller reported that during the epidemic of poliomyelitis in 1936-1937 there was a tendency for the disease to localize in the cerebrum and cerebellum. Peters now reports the pathologic changes found in 39 per cent of the patients. The cerebrum and cerebellum were involved in all those examined, and in some the brain showed more extensive changes than the cord. In fact, there was no part of the gray matter which failed to show changes in the ganglion cells and glial infiltration. The cerebral involvement has been more frequent in recent years than previously.

BRAHDY, Mount Vernon, N. Y.

PROPHYLAXIS OF SCARLET FEVER IN INSTITUTIONS. W. BRENNER, Ztschr. f. Kinderh. **60**:243 (Oct. 15) 1938.

Brenner calls Gabritschewsky the father of prophylactic inoculation against scarlet fever. In 1905 this worker inoculated man with a vaccine prepared from hemolytic streptococci cultured from patients with scarlet fever. Russian workers reported good results with his method.

Brenner inoculated 584 children with streptococcus vaccine between Jan. 1, 1937 and Jan. 7, 1938. Only 3 of these children (about 0.16 per cent) contracted scarlet fever and about 0.6 per cent contracted angina. During a similar period, 1935 to 1936, when vaccine was not used, 15 children (about 0.9 per cent) contracted scarlet fever and 39 (about 3.7 per cent) angina. Protection, which, according to the laws of immunity, should develop slowly, developed surprisingly early. In the majority of children giving the positive reactions to the Dick test the reactions were negative two to four days after injection. The early development of immunity finds support in experimental work reported in 1921 and 1937. Brenner discusses the possible nature of the "Frühschutz" (early protection).

SIEMSEN, Chicago.

SCARLET FEVER HEART. P. KISS and O. MALAGUZZI-VALERI, Orvosi hetil. **82**: 414 (April 30); 446 (May 7) 1938.

Under the title "Scarlet Fever Heart" the authors described the following symptoms: lability of the pulse rate, sometimes a systolic murmur, sometimes dilatation of the heart and sometimes arrhythmia. Lederer and Stollte believe that these symptoms are connected with variation in the body weight. If the body

weight diminishes, the symptoms are more noticeable. Kiss and Malaguzzi-Valeri examined the variation in the body weight during scarlet fever and the variation in the aforementioned symptoms but could not find any correlation between them. They examined also the correlation between anemia in scarlet fever and the symptoms of "scarlet fever heart," but even here they could not find any connection.

GÖTTCHE, Budapest Hungary.

DEHYDRATING THERAPY IN CASES OF INFANTILE PARALYSIS. B. VÁSÁRHELYI, Orvosi hetil. **82**:639 (June 25) 1938.

In twelve cases of infantile paralysis 20 to 40 cc. of 40 per cent dextrose was given intravenously daily for at least twelve days after the onset and every third hour a spoonful of 10 per cent sodium acetate. Besides this, the author gave 10 to 20 cc. of parents' blood or 10 to 20 cc. of convalescent serum intramuscularly. Vásárhelyi believes that this dehydrating method diminishes the pressure on the cells of the cord and hinders their destruction. GÖTTCHE, Budapest, Hungary.

EXTRASYSTOLIC ARRHYTHMIA IN SCARLET FEVER. P. KISS and J. ROMHÁNYI, Orvosi hetil. **82**:784 (Aug. 6) 1938.

Kiss and Romhányi report 2 cases in which myocarditis developed during scarlet fever. The myocarditis could be demonstrated by extrasystoles of ventricular origin. These extrasystoles disappeared later. GÖTTCHE, Budapest, Hungary.

HEART BLOCK IN SCARLET FEVER. P. KISS and R. MARTYN, Orvosi hetil. **82**: 946 (Sept. 24) 1938.

A 10 year old boy contracted scarlet fever in a typical form. The course was mild, but in the fifth week cervical adenitis and otitis media developed. Up to this time the heart did not show any pathologic changes; the pulse rate, however, was a little rapid (140 per minute). In the sixth week arrhythmia suddenly developed, and the electrocardiogram showed atrioventricular dissociation (type Wenckebach). The arrhythmia disappeared in twenty-four hours, but the electrocardiographic alterations remained constant for two weeks and then disappeared. Kiss and Martyn think that these symptoms were caused by focal infiltration next the atrioventricular leading system and that after absorption of the focus the trouble ceased.

GÖTTCHE, Budapest, Hungary.

Acute Infectious Diseases

THE RHEUMATIC SUBCUTANEOUS NODULE AND SIMULATING LESIONS. H. KEIL, Medicine **17**:261 (Sept.) 1938.

Keil reviews the early descriptions of rheumatic nodules and discusses the differential diagnosis of such nodules.

The typical rheumatic nodule is described as subcutaneous and covered by intact skin; it is not attached to the skin but to the deeper structures. They are more common with severe rheumatic fever and are almost always indicative of cardiac involvement. They are also more common with the more severe degrees of cardiac involvement—aortic lesions and pericarditis. The gravity of the prognosis roughly parallels the number and magnitude of the lesions. Rheumatic children exhibiting subcutaneous nodules have rarely been recorded as having lived to maturity.

The similarities and differences between subcutaneous nodules and Aschoff bodies are discussed. The former are macroscopically visible and palpable; the latter are microscopic. Necrobiosis occurs more commonly in the former than in the latter. The subcutaneous lesion is exudative; that in the heart is proliferative. Multinuclear cells occur more often in the Aschoff body. This heals by scar formation; the subcutaneous nodule, sometimes by calcification. Supravital stains

indicate considerable similarity between rheumatic nodules and the nodules of rheumatoid arthritis, and the author concludes that histologically the rheumatic nodule is not a specific lesion, with the possible exception of infiltrations in the galea aponeurotica, and that its recognition should be based on clinical criteria.

LIMPER, Louisville, Ky.

STAPHYLOCOCCAL INFECTIONS AT SINGAPORE. W. HUGHES, Quart. J. Med. **7**:537 (Oct.) 1938.

Hughes presents a clinical and to a less extent an immunologic discussion of staphylococcic infections.

One interesting part of the discussion deals with scalp infections in Chinese children. The incidence of this condition may be indicated by the citation of census figures from the General Hospital, Singapore. During the month from July 12 to August 12, 1937, 170 patients were admitted to the infants' ward (1½ years of age and under). In one ward there were 63 infants, 22 of whom had boils on their scalps.

Since the Chinese discarded the habit of wearing pigtails, they often go to the other extreme of shaving or closely clipping the hair from the head. The author thinks that small abrasions resulting from this procedure may constitute sites in which infections start.

The mechanism of the spread of staphylococcic infection is also discussed.

BRADFORD, Rochester, N. Y.

"Q" FEVER: FACTORS AFFECTING THE APPEARANCE OF RICKETTSIAE IN MICE. F. M. BURNET and M. FREEMAN, M. J. Australia **2**:1114 (Dec. 31) 1938.

From their experiments Burnet and Freeman conclude that the best method of using mice for the isolation of the "Q" fever rickettsias from human blood or other suspected material is to inoculate 6 or 8 mice intraperitoneally. Two should be killed on the tenth and fourteenth days after inoculation, and smears of the spleen and liver should be examined. The remainder should be kept for three to four weeks and then bled for serum. Positive agglutination with an emulsion of the rickettsias will provide final evidence that the mice were specifically infected.

GONCE, Madison, Wis.

BLACKWATER FEVER FOLLOWING THE ADMINISTRATION OF "ATEBRIN." C. E. M. GUNTHER, M. J. Australia **2**:1119 (Dec. 31) 1938.

Gunther reports a case in which atebrin (a derivative of acridine) initiated blackwater fever in a patient with latent malaria.

GONCE, Madison, Wis.

NOTE ON A SERIES OF LABORATORY INFECTIONS WITH THE RICKETTSIA OF "Q" FEVER. F. M. BURNETT and M. FREEMAN, M. J. Australia **1**:11 (Jan. 7) 1939.

Burnett and Freeman describe 5 laboratory cases of mild or subclinical "Q" fever. The infections were recognized by serologic tests—by rickettsial agglutination and by protection tests in mice. A mite, Liponyssus bacoti Hirst, is suspected of being the responsible vector from experimentally infected mice.

GONCE, Madison, Wis.

A FURTHER SERIES OF LABORATORY INFECTIONS WITH THE RICKETTSIA OF "Q" FEVER. D. J. W. SMITH, H. E. BROWN and E. H. DERRICK, M. J. Australia **1**:13 (Jan. 7) 1939.

Smith, Brown and Derrick describe 2 laboratory cases of acute "Q" fever. In each case the "Q" virus was shown to be present in the patient's blood by guinea pig inoculation, and "Q" agglutinins were demonstrated to be present in

the serum during convalescence. The infections were associated with experimentation on mice. The mite Liponyssus bursa (Berlese) was present at times on the mice and was therefore a potential vector, but the infections are ascribed to direct contact with infected mouse tissues.

GONCE, Madison, Wis.

DIAGNOSIS OF KALA-AZAR BY STERNAL PUNCTURE. S. BARTSOCAS, Arch. de méd. d. enf. **42**:153 (March) 1939.

The diagnosis of kala-azar is rendered difficult by the fact that the number of organisms appearing in the blood is small. The only practical method of demonstrating the parasite thus far found has been puncture of the spleen, which involves certain risks of hemorrhage and splenic lacerations. Aspiration of the liver is likewise objectionable, and puncture of the inguinal glands is not satisfactory.

In 1928 Arinkine recommended sternal puncture for more certain diagnosis. The author has employed this method in 24 cases, with positive results in each.

AMESSE, Denver.

TREATMENT OF TRICHINOSIS WITH CALCIUM. E. SOGEMEIER, München. med. Wchnschr. **85**:699 (May 6) 1938.

Sogemeier, of San Mateo, Calif., reports 3 cases in which severe trichinosis was successfully treated by intravenous injections of calcium gluconate. The patients felt improved after the first injections. They received 5 cc. of calcium gluconate daily. The fever subsided after the first injection in 1 patient and after the third injection in the other 2. This treatment was suggested to the author when he read the report by Gilbert and Stewart of the successful use of calcium in the treatment of a patient bitten by a black widow spider.

BRAHDY, Mount Vernon, N. Y.

CHANGES IN THE BODIES OF THE SPINAL VERTEBRAE DUE TO TETANUS. L. POKORNY, Röntgenpraxis **9**:813 (Dec.) 1937.

It was first recognized during the World War that the violent muscular spasms of tetanus are capable of producing roentgenographically demonstrable deformity of the vertebral bodies. This observation has been confirmed repeatedly in recent years. The phenomenon has been reported to occur in between 20 and 100 per cent of cases of tetanus. Roughly, the following are the diagnostic features:

1. Flattening of the bodies of one or more vertebrae. This varies in degree, sometimes being marked enough to result in serious dwarfing of the body length.
2. Planes of condensation along the cephalad and caudad margins of the compressed vertebrae.
3. Absence of evidence of foci of infection.
4. Preservation of the integrity of the intervertebral disks.
5. Absence of evidence of cold abscess.
6. Frequent occurrence of kyphosis.

The most obvious explanation of the mishap is found in the great force exerted by the spinal musculature during the tetanic convulsions; its occurrence does not require, as some have suspected, that the vertebral bodies be previously weakened by disease; nor is it necessary that the tetanus be of unusually long duration.

An illustrative case history is narrated. The patient was a previously healthy 10 year old boy. A roentgen examination was made because of spinal curvature following tetanus. One of the editors of *Röntgenpraxis* adds brief notes on a second case in which the patient was a child of 11 years.

No information is given concerning prevention, treatment or prognosis.

MCCUNE, New York.

Otologic Relations to Dysentery in Infancy. B. Galcsik, Orvosi hetil. **82**:128 (Feb. 5) 1938.

In every case of dysentery an otoscopic examination was made, and if mastoidal empyema was suspected an operation was immediately made, with the infant under local anesthesia. This antrotomy was made even in cases in which the otoscopic examination did not show pathologic changes. In very few cases was a healthy antrum found. Up to the years 1930-1932 these operations were rarely performed, and the mortality was 42 to 45 per cent. From 1933 on, when this operation became the rule, the mortality fell to 22 per cent. Galcsik thinks that in this great improvement, apart from the progress in internal treatment, intensive otologic treatment plays a great role.

Göttche, Budapest, Hungary.

Loss of Weight Caused by Protozoa. P. Véghelyi, Orvosi hetil. **82**:321 (April 2) 1938.

Véghelyi examined 1,500 children and found that those children who had parasites in their stools were for the most part thinner than they should be, whereas their height was normal. Of the children who had Giardia in their stools, 85 per cent were below normal weight; 21 of them were treated with acetarsone (stovarsol), and Giardia disappeared from the stools of 19; all these 19 children began to gain weight. In 2 cases Giardia did not disappear, and the children did not gain. Endamoeba histolytica was found in the stools of 26 children, and 24 of these were below normal weight; thus far 5 patients have recovered, and all these 5 children have gained weight. Seven children were infected with Chilomastix; none of them reached normal weight. The author thinks that in many children the condition of being underweight is caused by these protozoa and that the loss can be stopped by demolition of the protozoa. It seems that these parasites are not so innocent as was believed heretofore.

Göttche, Budapest, Hungary.

Treatment of Rheumatism in Childhood. B. Horváth, Orvosi hetil. **82**:651 (July 2) 1938.

In Hungary rheumatism is not so widespread as in the north and west of Europe. In the last twenty years only 0.75 per cent of the illnesses at the Budapest child clinic were rheumatic diseases. According to the report of the child clinic at Szeged, the number of rheumatic diseases shows a steady increase. In the last three to four years five times as many children were treated for rheumatism as formerly. In most cases, of course, heart involvements played the greatest role. Besides chemical treatment, balneotherapy and mechanical treatment of the joints to hinder shrinking and distortion are very important.

Göttsche, Budapest, Hungary.

Amebic Dysentery in Childhood. P. Véghelyi, Orvosi hetil. **82**:853 (Aug. 27) 1938.

Endamoeba histolytica occurs also in Hungary. It is found in 2 to 6 per cent of the patients of the child clinic in Budapest. These infections can cause various clinical pictures. One can see sometimes the clinical picture of chronic colitis; one can sometimes find the clinical picture of acute bacillary dysentery, even sometimes the symptoms of chronic appendicitis. The best treatment is the use of chiniofon, which in most of the patients leads to prompt healing.

Göttsche, Budapest, Hungary.

Septic Illness of a Young Infant Caused by Salmonella suipestifer Bacilli. I. Gagyi and Pomóthy, Orvosi hetil. **82**:1155 (Nov. 26) 1938.

An 8 week old infant was admitted to the child clinic at Pécs with septic fever and neurologic symptoms of toxic origin. The blood culture showed bacilli which

were like Bacillus paratyphosus B, but further studies showed that the organism was Salmonella suipestifer. It is interesting that the illness ran its course without localization and that the infant recovered. GÖTTSCHE, Budapest, Hungary.

RAT BITE FEVER IN CHILDREN. E. YOKOTE, Acta pædiat. japon. **44**:853 (June) 1938.

Ten cases of rat bite fever in children ranging in age from 1 month to 4 years and 2 months are reported. The primary lesion was either on an extremity or on the face. The incubation period was from six days to one month. In 50 per cent of these cases the fever showed typical curves, and the urticarial eruptions usually coincided with the rise in temperature. Regional lymphadenopathy was the rule. No constant blood picture was associated with the disease, although in most cases anemia was present. In only a single case was the Wassermann test positive. Arsenicals were effective in all cases. KATO, Chicago.

BACILLARY DYSENTERY IN YOUNG INFANTS. B. LAMMERTS VAN BUEREN and J. H. DE HAAS, Maandschr. v. kindergeneesk. **7**:461 (Sept.) 1938.

This is a description of bacillary dysentery during the first three months of life as observed in 11 infants. For the diagnosis at this age, microscopic examination of the feces is necessary. Alimentary treatment is recommended.

VAN CREVELD, Amsterdam, Netherlands.

MENINGEAL SYMPTOMS CAUSED BY ASCARIS. J. C. WIJCKERHELD BISDOM, Maandschr. v. kindergeneesk. **7**:490 (Sept.) 1938.

A typical picture of verminal meningitis was observed in a girl of 6 years.

VAN CREVELD, Amsterdam, Netherlands.

TYPHOID CARRIERS. H. PEETERS and A. C. RUYS, Nederl. tijdschr. v. geneesk. **82**:3741 (July 30) 1938.

Epidemiologic investigations of typhoid require close cooperation of the epidemiologist and the bacteriologist. By refined bacteriologic diagnostic measures, among others, by the Wilson-Blair culture medium—typhoid carriers may be detected who otherwise would remain undiscovered for a long time. In Amsterdam and its environs, on systematic epidemiologic examination 50 typhoid carriers have been found. Of these 4 have been cured by cholecystectomy; 6 after a shorter or longer time ceased to produce bacteria, and 6 have died. Of the remaining 34, 17 are being examined regularly; the other 17 refused examination or have left the city.

The ways in which the various carriers have been detected are described—for example, they have been detected by the number of infections they have caused. With some few exceptions, the details of which are given, these persons, after having been recognized as carriers have not been the source of any further infections. VAN CREVELD, Amsterdam, Netherlands.

TYPHOID AT VOLENDAM, NETHERLANDS. H. PEETERS and A. C. RUYS, Nederl. tijdschr. v. geneesk. **82**:4171 (Aug. 27) 1938.

At Volendam typhoid prevails endemically, mostly among persons under the age of 20 years. For the spread of the organisms the water of the numerous canals is of much importance. The authors could repeatedly grow typhoid bacteria from the water. VAN CREVELD, Amsterdam, Netherlands.

CLINICAL OBSERVATIONS IN ACUTE RHEUMATIC FEVER. II. P. H. KRAMER, Nederl. tijdschr. v. geneesk. **82**:5199 (Oct. 22) 1938.

Kramer discusses rheumatic chorea in young persons of 14 to 20 years and calls attention to the fact that at this age the course of the disease is more serious than in childhood, especially with regard to the psychic symptoms. In comparing the symptoms of involvement of the brain in acute rheumatic infections at different ages, Kramer regards this severe chorea of adolescence clinically as a transition of the benign chorea of childhood to the hyperpyretic form of acute rheumatism in adults.

VAN CREVELD, Amsterdam, Netherlands.

THERAPY OF TETANUS. A. VAN MEETEREN, Nederl. tijdschr. v. geneesk. **82**:5658 (Nov. 26) 1938.

A boy of 17 years was cured of tetanus by large doses of serum and intravenous injections of 33.3 per cent alcohol.

VAN CREVELD, Amsterdam, Netherlands.

Chronic Infectious Diseases

ANTITUBERCULOUS VACCINATION WITH BCG. L. NEGRE, Union méd. du Canada **67**:1147 (Nov.) 1938.

Negre states emphatically that BCG is harmless and incapable of regaining its virulence. Injected into laboratory animals by various routes, it sensitizes them to tuberculin and produces small follicular lesions which never caseate.

DAFFINEE, Boston.

TYPES OF PULMONARY TUBERCULOSIS—CREEPING TUBERCULOSIS. L. S. T. BURRELL, Brit. J. Tuberc. **32**:65 (April) 1938.

The type of infection referred to occurs in families who include a member with active tuberculosis. Other members of the family may become infected to varying degrees. The infection in certain ones is such as to produce lesions but not to cause toxic or other symptoms. In such a group roentgen examination may reveal advanced disease in a subject apparently in normal health. No treatment appears to have any effect, and the progress is usually downward. Finally there is a sudden breakdown, and the patient dies of miliary tuberculosis or bronchopneumonia. The progress of such a case is described.

EUGENE H. SMITH, Ogden, Utah.

CONTACTS WITH SPUTUM POSITIVE TUBERCULOUS CASES. E. WILLIAMS, Tubercle **19**:495 (Aug.) 1938.

The incidence of tuberculous infection is unusually high among children who are exposed in their homes to persons whose sputum is positive for tubercle bacilli. Graded Mantoux tests on 68 such children in Cardiff, Wales, were positive in 87 per cent. Among children under 5 years, 66 per cent reacted to the highest dilutions of tuberculin. Of infected members of the family, the mother was the greatest source of danger; not one single child in contact with an infected mother escaped. Infected sisters were next on the danger list.

EUGENE H. SMITH, Ogden, Utah.

DEATH FROM HAEMOPTYSIS OCCURRING IN AN INFANT OF NINE MONTHS. C. O'LEARY, Tubercle **19**:513 (Aug.) 1938.

When 3 months of age, an infant was examined as a "contact." A roentgenogram revealed the presence of some "haziness in the left perihilar region." Later bronchitis and pneumonia developed. Roentgenograms showed "a few nodes in

the upper part of the left perihilar region and also in the lower part of the right perihilar region." The Mantoux test was performed; there was a positive reaction to a 1:1,000 dilution of old tuberculin. The child died suddenly when 9 months old, from hemorrhage. At postmortem examination a tuberculous infiltration was found in the upper and lower lobes of the left lung. In the lower lobe, in the region of the hilus there was an area of denser infiltration. Section through this area showed a central excavation with erosion into a large blood vessel which had led to the fatal hemorrhage. EUGENE H. SMITH, Ogden, Utah.

PRECOCIOUS SEXUAL DEVELOPMENT. J. LHERMITTE, Rev. neurol. **69**:380 (April) 1938.

In a boy of 11 years macrogenitosomia was associated with hydrocephalus. In early infancy the head was smaller than normal. The child did not walk until the age of 4 years and talked late. Convulsions were present until the fourth year. Following a hard fall, in which the skull was fractured, the head began to increase in size. Two years later genital hypertrophy was noted. Lhermitte classifies this case as an instance of Pellizzi's syndrome. DAFFINEE, Boston.

PRIMARY TUBERCULOSIS OF MUSCLE. S. KUSHEZAKI and K. SAITO, Beitr. z. klin. Chir. **165**:177, 1937.

Tuberculosis of skeletal muscle is very rare—less than 100 cases have been reported. It occurs more frequently in the muscles of the extremities than in those of the trunk. In order of frequency the muscles involved are those of: forearm, upper part of arm, lower part of leg and thigh. A single muscle is involved usually; more than one, very seldom. The disease is found most frequently in males—the ratio is 2:1. In some cases it is initiated by trauma or straining and is not dependent on a primary tuberculous condition. In more than half the cases reported there was abscess formation; in others, a tuberculous nodule or sclerotic myositis. In most of the cases examination shows small subcutaneous tumors, most evident on contraction of the muscle—freely movable and tender. The overlying skin is always normal. It is very difficult to make a clinical diagnosis—histologic or bacteriologic investigations are necessary. In order to get ideal healing, radical removal of affected tissue is absolutely necessary. Function can usually be restored in this way, whereas it may not be with conservative treatment.

The authors report 2 cases. In one the right pectoralis major muscle was involved and in the other the muscles of all the extremities were affected symmetrically. The first patient was a man 24 years of age in whom no primary tuberculous lesion was found. In the second the patient was a man 34 years of age with calcified hilar nodes revealed in the roentgenogram. The condition in both was of the most infrequent type—myositic. Both patients were operated on and then were given roentgen therapy. Function was restored. RUBIN, Boston.

END RESULTS IN PATIENTS WITH SEVERE TUBERCULOSIS OF BONE AND WITH LARGE TUBERCULOUS SEQUESTRAE. H. NASE, Beitr. z. klin. Chir. **165**:572, 1937.

It is important to consider the tuberculous patient as a whole and to realize what the human organism can do to help.

Single involved areas in the small bones of the extremities are better handled very conservatively, instead of risking bad results and chances of injury to the epiphyses in children with operative procedures. In doubtful cases the upper extremities show better chances of healing than do the lower extremities if treated conservatively. Otherwise, operation should be performed whenever there is a good possibility of reaching the focus so as not to cause damage.

RUBIN, Boston.

THE PROBLEM OF TUBERCULOSIS IN INFANTS IN RELATION TO MODERN INVESTIGATIONS OF THE ULTRAVIRUS. TOPIC III. THE FOURTH INTERNATIONAL PEDIATRIC CONGRESS, ROME, SEPTEMBER 1937. Acta pædiat. **22**:168, 1938.

Dufourt, of Lyon, presents a lengthy review of the studies that led up to the concept of the ultravirus of tuberculosis. Cohen, of Brussels, analyzes critically the role of this somewhat hypothetic factor in infantile infections. Several Italian investigators supply descriptions of their experience. It is of interest that no German or English voice was raised in the discussion of this topic.

McCUNE, New York.

THE CONTAGIOUSNESS OF INFANTILE TUBERCULOSIS. TOPIC III, B. THE FOURTH INTERNATIONAL PEDIATRIC CONGRESS, ROME, SEPTEMBER 1937. Acta pædiat. **22**:229, 1938.

Wallgren admits that tuberculous children who cough violently can disseminate tubercle bacilli; however, he feels that the practical danger of cross infections arising in this way is numerically small in comparison with the menace of patients with caseating tuberculosis and of infected cattle. Hence, he does not think it necessary to isolate from their tuberculin-negative comrades children harboring benign primary tuberculous infections. Capon, of Liverpool, reports conclusions based on a survey of the incidence and manifestations of tuberculosis in 200 children; his results do not differ widely from the results of similar surveys in this country. Herlitz emphasizes the danger represented by infected dogs and cats. The general sense of the speakers appears to be that infants and children can transmit tuberculous infection but that the spread from this source is relatively small.

McCUNE, New York.

PROBLEMS OF PREVENTION AND TREATMENT IN JUVENILE TUBERCULOSIS. TOPIC III, C. THE FOURTH INTERNATIONAL PEDIATRIC CONGRESS, ROME, SEPTEMBER 1937. Acta pædiat. **22**:277, 1938.

Administrative and medical prophylactic measures are discussed by representatives of the European and American schools of thought. The present status of vaccines is reviewed. Therapeutically, open air treatment and artificial pneumothorax receive the most attention. In the appendix are assembled miscellaneous contributions bearing on divers aspects of tuberculous infection in children.

McCUNE, New York.

TABES DORSALIS IN CHILDREN. S. WENDT, Acta pædiat. **23**:245, 1938.

Aside from the large number of cases of diffuse cerebrospinal syphilis in children that have been reported in the literature there are on record approximately 140 authentic cases of tabes dorsalis occurring before puberty. Wendt adds 2 cases to this collection. The first patient was a 13 year old boy, the scion of a family known to be syphilitic. There was a positive Wassermann reaction of the blood and of the cerebrospinal fluid; the pupillary reaction to light in one eye was diminished with reaction during accommodation preserved bilaterally; there was absence of tendon jerks; there were girdle pains, aphasia, abdominal paralysis and disturbance of cremasteric reflexes, together with a Babinski phenomenon. Treatment was undertaken with an inoculation of malaria organisms. The results of the therapy are not described. The second patient, a girl of 9 years, exhibited left-sided ptosis, Argyll Robertson pupils and anisocoria, Hutchinson's teeth and absence of some of the deep reflexes. The Wassermann reaction of the blood and of the spinal fluid was positive. The spinal fluid showed increased protein and moderate pleocytosis. Subsequently the extrinsic muscles of the left eye became paralyzed, and diplopia developed. Malaria treatment was given, with striking improvement of the ocular abnormalities and with return of the cerebrospinal

fluid to normal. Because of a residual positive Wassermann reaction of the blood and of the spinal fluid, treatment with mercury compounds and arsphenamine was started. During the course of treatment bilateral interstitial keratitis appeared, with resultant diminution of visual acuity. Her status at the time of the report was not altogether gratifying. The differential diagnosis of juvenile tabes dorsalis and its treatment are discussed; the state and results of the therapy are still unsettled.

McCune, New York.

Diseases of Blood, Heart and Blood Vessels and Spleen

Complete Transposition of the Great Vessels. H. B. Taussig, Am. Heart J. **16**:727 (Dec.) 1938.

Clinical studies were made on 4 patients with complete transposition of the great vessels. The roentgenographic findings were sufficiently characteristic to establish the diagnosis during life. Owing to the altered position of the great vessels relative to the ventricles, a characteristic change in the cardiac silhouette was observed. When the patient was placed in the left anterior oblique position, there was an increase in the width of the roentgenographic shadow cast by the great vessels. This finding is pathognomonic of the condition. Other findings were cardiac enlargement, especially of the right ventricle, and a narrow aortic shadow in the anteroposterior roentgenogram.

Gibson, Chicago.

Normal Antithrombin of the Blood and Its Relation to Heparin. A. J. Quick, Am. J. Physiol. **123**:712 (Sept.) 1938.

The experiments presented suggest that the normal antithrombin of the blood is albumin or a constituent closely associated with that fraction. Since fibrinogen has a greater affinity for thrombin than albumin, no appreciable neutralization or inactivation of thrombin occurs until all the fibrinogen is converted to fibrin. By adding heparin to albumin, the affinity of the latter for thrombin is so increased that the thrombin will be combined with the heparin-albumin complex before it can react with fibrinogen. When albumin is heated to 67 C. it loses its ability to neutralize thrombin, and the addition of heparin no longer produces any antithrombin action, which indicates that heparin merely intensifies the inherent antithrombin power of albumin and is itself not an antithrombin.

Nourse, Cleveland.

Preparation and Properties of the Charcoal Adsorbate of Liver Extract. J. Sladek and J. Kyer, Proc. Soc. Exper. Biol. & Med. **39**:227 (Nov.) 1938.

A concentrate of liver extract is prepared by precipitating the protein by the formation of calcium carbonate through the addition of 27.5 Gm. of anhydrous calcium chloride and then 26.5 Gm. of anhydrous sodium carbonate to the aqueous filtrate after alcoholic extraction of 5 pounds (2,268 Gm.) of fresh liver. The active principle is subsequently adsorbed on norite (a specially activated plant charcoal). From 100 Gm. of fresh liver 3 cc. of extract is obtained. This 3 cc. of extract is capable of increasing the reticulocyte count in the patient with pernicious anemia. The extract is effective in the treatment of patients with this disease and is able to maintain the red cell count at normal levels. Tyrosine is present in the final product.

Hansen, Minneapolis.

Hand-Schüller-Christian Disease of Scapula. W. C. Wilson, Tr. Med.-Chir. Soc. Edinburgh, 1938, p. 185; in Edinburgh M. J., October 1938.

A boy of about 2½ years was admitted to the hospital in September 1936 with a swelling over the right scapula which roentgen examination indicated was a

calcified tumor of the scapula. The scapula was not excised but was treated with roentgen radiation. The tumor disappeared, and eighteen months later there was no sign of recurrence. The biopsy showed that the tumor contained many eosinophils, which in the absence of blood changes indicated Hand-Schüller-Christian disease. This illustrates the value of conservatively using roentgen radiation in the presence of a possibly inoperable osteogenic sarcoma.

NEFF, Kansas City, Mo.

HYPERTROPHY OF THE THYMUS IN THE COURSE OF ACUTE LEUKEMIAS OF CHILDHOOD. R. PIERRET, L. CHRISTIAENS and POPOFF, Arch. de méd. d. enf. **42**:5 (Jan.) 1939.

It is commonly understood that enlargement of the thymus is not included in the syndrome of acute leukemia in any form, and in clinical studies the thymus is considered entirely apart from lymphoid structures. A number of scattered observations, however, seem to prove that this view is not in strict accordance with fact. In some instances the report relates to a tumor of the anterior mediastinum more or less associated with the thymus and complicated at a later stage with acute leukemia of either the lymphoblastic or the monoblastic type. Further instances are described wherein during the course of leukemia there is frank hypertrophy of the thymus, demonstrated either clinically or at autopsy. Three such cases are reported in this contribution, with illustrations and an extended comment on the pathogenesis.

AMESSE, Denver.

ANEURYSM OF THE CUBITAL ARTERY IN AN INFANT OF NINE MONTHS. M. FÈVRE and A. BOHN, Arch. de méd. d. enf. **42**:97 (Feb.) 1939.

Arterial aneurysm is extremely rare in childhood, particularly in nurslings. The condition is reported in a male infant of 9 months with a history of anorexia and malnutrition shortly after birth, though at the time of the appearance of a small tumor on the palmar surface of the left wrist the infant seemed in excellent health. At first believed to be a synovial cyst, this abnormality soon assumed all the characteristics of an aneurysm involving the cubital artery. With the imminent danger of rupture in mind, surgical intervention was considered advisable, and the sac was dissected from the neighboring tissues. Complete recovery followed.

Syphilis and tuberculosis having been excluded, the authors conclude that the origin of the aneurysm was traumatic.

AMESSE, Denver.

MULTIPLE PREGNANCIES IN A WOMAN WITH CONGENITAL CYANOSIS DUE TO HEART DISEASE. BROUSTEL, LÉVY and DIARD, Bull. Soc. d'obst. et de gynéc. **27**:759 (Dec.) 1938.

A Spanish woman 37 years of age had been cyanotic since birth because of congenital heart disease. A thorough study of her heart resulted in a diagnosis of pulmonary stenosis and insufficiency. She came to the clinic because the cyanosis and dyspnea had become worse in the past six years. Her menstrual periods had always been normal. At the age of 28 years she delivered a normal full term infant, which survived. At the age of 30 years and again at the age of 32 years she delivered a dead, macerated fetus at term. Her fourth and last pregnancy occurred at the age of 34 years and resulted in a normal full term infant, which survived. There was no exacerbation of the cardiac symptoms during any pregnancy. She showed no evidence of syphilis. The authors believe the mother's cyanosis was responsible for the intrauterine death of the 2 infants. They offer no explanation, however, of why 2 survived under the same conditions.

GLASER, Rochester, N. Y.

CONGENITAL CARDIOPATHY WITHOUT MURMURS. E. FILGUEIRAS and A. FIALHO, Rev. franç. de pédiat. **13**:192, 1937.

A girl had showed marked cyanosis and dyspnea since birth. There was no marked enlargement of the heart; the tones were regular and clear, and no murmurs were detected at any time. The child died at the age of 2 years. Examination of the heart showed auricular and ventricular septal defects and the aorta arising from the right ventricle. The pulmonary artery originated as a blind sac but was joined shortly after its origin by a communicating vessel from the aorta. The embryologic aspects are discussed. The article is illustrated.

LESLIE, Evanston, Ill.

BIGEMINAL PULSE. A. PEGLUI, Arch. argent. de pediat. **2**:299 (Sept.) 1938.

A case of bigeminal pulse of seven years' duration is described. The patient was a 9 year old girl. The presence of this anomaly was confirmed by electrocardiographic tracings. Roentgenograms revealed a small heart with dilatation of the right cavities. The third heart sound was accentuated; otherwise there were no definite clinical findings. The child is being followed up.

SCHLUTZ, Chicago.

ACUTE LEUKEMIA (HEMOCYTOBLASTIC). O. ORTIZ MACHADO, Bol. coll. med. Camaguey **1**:29 (Oct.-Dec.) 1938.

Three forms of acute leukemia are recognized after the classification of Ferrata, namely: (1) acute myelosis, with leukemic, aleukemic and erythroleukemic forms; (2) acute lymphadenosis, with leukemic and aleukemic forms, and (3) a hemoblastic syndrome. A short review of the leukemias is presented, with a report of leukemia in a girl aged 10 years. In this case the total leukocyte count varied between 1,900 and 2,400 per cubic millimeters, with lymphocytes 74 to 83 per cent and immature forms 5 to 20 per cent. The immature cells are the primitive cells of the bone marrow—hemocytoblasts. The diagnosis was leukemia with hemocytoblastosis, leukopenic in type.

DWYER, Kansas City, Mo.

VITAMIN C THERAPY FOR HEMORRHAGIC DIATHESES. H. WINCKELMANN, Med. Klin. **34**:906 (July 8) 1938.

In the case reported vitamin C therapy was ineffective, but the main object of the article is to emphasize that vitamin C therapy can be regarded as successful or unsuccessful only when the body's deficit of vitamin C has been corrected. At times very high doses are necessary.

SIEMSEN, Chicago.

XANTHELASMATOSIS IN CHILDHOOD. K. SOEHRING, Monatschr. f. Kinderh. **77**:315 (March) 1939.

Soehring reports 2 cases with photographs. He prefers the term "xanthelasmatosis" to "xanthoma", which, he believes indicates a condition that has a slightly different histologic picture. No improvement followed a cholesterol-free diet.

GERSTLEY, Chicago.

EOSINOPHILIA IN PREMATURE INFANTS. C. DIECK, Monatschr. f. Kinderh. **78**:64 (March) 1939.

A study was made of 21 premature infants. Fourteen of them showed eosinophilia beginning in the second or third week and reaching its maximum at the sixteenth week. In absolute count they reached a maximum of 2,400 per cubic millimeter, and in percentage, a maximum of 22.

GERSTLEY, Chicago.

MYELOGENOUS CHLOROMA. A. A. LEBEDIEV, Vestnik oftal. **11**:195, 1937.

Lebediev states that this is the eighty-first case of myelogenous chloroma reported in the literature. A boy aged 13 years presented bilateral exophthalmos with bilateral papilledema, more marked on the right side. There were paralysis of the facial nerve on the right side, lagopththalmos and superficial keratitis in the right eye. An immovable firm tumor could be palpated at the outer upper third of the orbit. The blood picture indicated a disease of the blood-forming organs, suggesting neutrophilic leukocytosis. Autopsy showed chloroma in each orbit, numerous metastases in the occipital and temporal bones and in the aponeurosis and intercostal muscles and hyperplasia of the brain. The growths were greenish. There was general hyperplastic lymphadenitis, the lymph nodes having a greenish color on cross section. The clinical differential diagnosis, treatment and prognosis are discussed.

The conclusions are are follows:

1. Chloroma belongs to one of the forms of leukemia.

2. The diagnosis of chloroma in some cases is difficult, particularly in the absence of the characteristic color.

3. Roentgen examination does not always aid in making the diagnosis.

4. Biopsy is valuable for early diagnosis.

5. Roentgen therapy is the only logical treatment at present.

6. Each case of chloroma should be reported in order that more material may be obtained for studies of the causes, early diagnosis and treatment of the disease.

SITCHEVSKA. [ARCH. OPHTH.]

HEMOSEDIMENTATION OF HEALTHY LACTATING WOMEN. Y. KOKUBO, Tohoku J. Exper. Med. **35**:361 (March 15) 1939.

From the result of an investigation on 339 lactating women some of whom yielded milk positive to the Arakawa reagent and others milk negative to this reagent, Kokubo reaches the following conclusions: 1. The sedimentation rate of the red blood corpuscles of normal lactating women shows a slight acceleration in most cases. 2. Apparently healthy lactating women who are secreting milk negative to Arakawa's reagent shows an increase in sedimentation rate, probably due to avitaminosis B_1.

LESLIE, Evanston, Ill.

Diseases of Nose, Throat and Ear

ACUTE LARYNGITIS IN INFANTS. G. J. GREENWOOD, Illinois M. J. **75**:52 (Jan.) 1939.

Following a review of the anatomy, histology, bacteriology, symptomatology and therapeutics of acute laryngitis in infants, with a discussion of 14 cases seen at the Children's Memorial Hospital during the winter of 1933-1934, Greenwood arrives at the following conclusions. Specific laryngitis in infants is a pertinent pediatric problem. When signs of obstructive dyspnea appear, the endoscopist or the endoscopically trained laryngologist should be summoned. For immediate relief intubation or tracheotomy is necessary. Greenwood believes that in an infant low tracheotomy conserves the laryngeal structure better than intubation.

BARBOUR, Peoria, Ill.

EPHEDRINE IN A PHYSIOLOGIC VEHICLE AND LATERAL HEAD LAW POSTURE IN TREATMENT OF THE NOSE AND SINUSES. S. N. PARKINSON, J. A. M. A. **112**:204 (Jan. 21) 1939.

As replacing many drugs for intranasal therapy and the Proetz position, Parkinson advocates lowering the head at a 45 degree angle in the lateral position

and instilling ephedrine in a physiologic vehicle for treatment of the common cold with a view to averting complications involving sinuses, ear and other parts. In this method all the ostia will be exposed to the solution, more will enter the throat but will be evacuated through the nose, and no damage will be done to the mucosa.

HEERSMA, Kalamazoo, Mich.

SINUSITIS IN CHILDREN. E. C. MITCHELL, J. A. M. A. **112**:207 (Jan. 21) 1939.

The treatment of sinusitis in children presents a favorable outlook for recovery and development of the sinuses if the condition is cared for early. Early recognition of the etiologic nature is important, especially if this is allergic. The treatment varies with the stage of the disease; it is outlined for the acute, subacute and chronic stages. The condition should be followed by roentgen examinations and observations for foci of infection.

HEERSMA, Kalamazoo, Mich.

PETROSQUAMOUS OSTEOMYELITIS IN INFANTS. A. LASKIEWICZ, Rev. de laryng. **59**:342 (April) 1938.

Three cases of osteomyelitis of the petrous apex in children are discussed, together, with 2 cases in adults. One child came to autopsy. The haversian canals microscopically revealed infected thrombi. This observation tends to confirm the present opinion that infection is propagated by way of the vascular system. The author reviews extensively the various current classifications of mastoid cells. The prominent clinical symptoms of osteomyelitis of the petrous apex are: parietal headache on the involved side; pain in the occiput and in the orbit; involvement of the fifth and sixth and more rarely the seventh, eighth, ninth and tenth cranial nerves and commonly edema in the zygomatic region, which may extend to the lids and the occiput. Serious attacks lead to meningitis. Progression seems to be along the carotid canal.

BATSON, Philadelphia. [ARCH. OTOLARYNG.]

VITAMIN C AND ACUTE INFECTIONS OF THE UPPER RESPIRATORY TRACT. H. PUMARINO, Rev. chilena de pediat. **9**:603 (July) 1938.

In several cases of acute bronchopulmonary infection vitamin C was given by injection with favorable results. From his observations Pumarino believes that the vitamin has a detoxifying action. He recommends its use in the treatment of acute infections of the respiratory tract.

SCHLUTZ, Chicago.

STRIDOR CONGENITUS. E. TRAUB, Ztschr. f. Kinderh. **60**:222 (Oct. 15) 1938.

According to Finkelstein, stridor may be congenital or acquired. Among causes for congenital stridor he lists unusual narrowness of the laryngeal aperature, recurrent paralysis, laryngeal papilloma, retrosternal goiter, thymic enlargement, tumor at the base of the tongue, microglossia and macroglossia. More recently he concurs in Thomsen's view that there is a lack of coordination of laryngeal movements, central in origin. The view of multiplicity of causes is shared by many authors. Traub thinks thymic enlargement is a very infrequent etiologic factor.

Lemariey and Sergent described the following laryngoscopic findings as classic: rolling in and backward from front and outside of the margins of the epiglottis and aryepiglottis; laxity of the epiglottis, and vibration of the aryepiglottic folds during inspiration, causing stridor.

As a rule, the prognosis is good. However, on the basis of his experience in the cases which he reports, Traub points out that the prognosis may occasionally be serious. Narrowness of the larynx apparently predisposes to inflammation

which in turn may cause enlargement of the mediastinal glands. Bronchopneumonia, atelectasis and pneumothorax may result from hypoventilation of the lungs.

SIEMSEN, Chicago.

DEMONSTRATION OF PNEUMOCOCCI IN THE THROATS OF NORMAL INFANTS AND OF INFANTS WITH PNEUMONIA OR INFECTION OF THE UPPER RESPIRATORY TRACT. S. NARASAKI, Acta pædiat. japon **44**:1036 (July) 1938.

Pneumococci were found in the throats of 9.2 per cent (summer) and 2.6 per cent (winter) of all normal infants. In infants suffering from grip the frequency was 16 per cent; in those with bronchitis, 29.2 per cent and in those with pneumonia, 43.3 per cent. These observations prove that in the progress of the disease the frequency of positive cultures increases. The majority of diplococci belonged to type IV. Pneumococci were found in the throats of infants under 1 year of age as frequently as in the throats of older children.

KATO, Chicago.

Diseases of Lungs, Pleura and Mediastinum

INTRAMUSCULAR ADMINISTRATION OF ANTI-PNEUMOCOCCAL SERUM IN INFANTS AND CHILDREN. L. KRAHULIK, V. RUDOMANSKI and G. CUNNINGHAM, Proc. Soc. Exper. Biol. & Med. **40**:18 (Jan.) 1939.

Twenty-four infants and children with pneumonia were given 60,000 to 200,000 units of horse or rabbit antipneumococcic serum intramuscularly on the first to the eighth day of the disease. In 7 infants there was a critical drop in temperature and symptoms in twelve to twenty-four hours, and in 3 more infants the temperature subsided by lysis in twenty-four to forty-eight hours. In 10 older children there was a critical drop in temperature in twelve to twenty-four hours, and in 4 others the temperature subsided by lysis in twenty-four to forty-eight hours. There were no deaths.

HANSEN, Minneapolis.

ACUTE LUNG CONDITIONS, WITH PARTICULAR REFERENCE TO THEIR TREATMENT IN INFANTS. A. W. FRANKLIN, Practitioner **142**:429 (April) 1939.

Infants and young children differ strikingly from adults in the problems of management presented by acute pulmonary conditions. The differences become marked as serotherapy and chemotherapy provide more exact methods of attack. In infants, particularly, the question is not how to treat lobar or disseminated pneumonia, bronchitis or acute asthma but how to manage, feed, rest and make comfortable a patient in acute respiratory distress, with, for example, a blocked nose, difficulty in sucking and cyanosis. The treatment is unavoidably symptomatic, for accurate diagnosis of the pathologic condition at the onset, when the therapeutic need is greatest, is impossible in most cases. The difficulty of obtaining sputum, with the consequent impossibility of identifying the organism and, of course, of typing the pneumococcus, has seriously impaired the utility of serotherapy, and diagnostic uncertainty makes the indications for chemotherapy vague and a true assessment of its value well nigh impossible.

GELSTON, San Francisco.

ADVANCES IN THE TREATMENT OF BRONCHOPNEUMONIA OF INFANTS. B. SORIA, Arch. de méd. d. enf. **42**:146 (March) 1939.

In spite of medical progress, bronchopneumonia continues to levy a very high mortality on children in the early months of life. Unlike what one observes of lobar pneumonia, the evolution is irregular and its course not self limited, since the process is not confined to a single foyer; these multiple foci of infection prevent compensation by deeper respiration, and cyanosis with exhaustion follows.

On the theory that the entire pathogenesis rests on a major disturbance of the acid-base equilibrium, Soria classifies bronchopneumonia clinically into pulmonary, cardiohepatic, gastrointestinal, nervous and toxic types. In the absence of specific treatment, hypertonic solutions, with 15 to 20 per cent dextrose, are given intramuscularly or intravenously; isotonic solutions are injected subcutaneously or by the peritoneal route. Under this management the death rate in Soria's series has been reduced one third.

AMESSE, Denver.

PNEUMOCOCCIC EMPYEMA IN THE CHILD AND ITS TREATMENT BY ETHYLHYDROCUPREINE HYDROCHLORIDE, PNEUMOTHORAX AND BLOOD TRANSFUSION. B. TASSOVATZ, Rev. franç. de pédiat. **14**:372, 1938.

Tassovatz discusses his experience in 31 cases of pneumococcic empyema. Aspiration of the pus was followed by lavage and the injection of ethylhydrocupreine hydrochloride. This drug is valuable not only in promoting sterilization of the pleural cavities but also in destroying fibrinous exudates and so preventing thickening of the pleura and adhesions. When any large amount of pus was to be removed, air was injected into the pleural cavity to avoid sudden changes in intrathoracic pressure. In some cases transfusions were given to improve the general condition of the patient. The author feels that without this particular method of treatment many of the patients would have had to have surgical drainage.

LESLIE, Evanston, Ill.

TRANSFUSION IN BRONCHOPNEUMONIA. A. CASTELLANOS and R. MONTERO, Arch. de med. inf. **8**:115 (April-June) 1939.

Castellanos and Montero believe that transfusion is of great benefit in bronchopneumonia, for two reasons: First benefit arises from colloid shock, and second, from passive immunization by the antibodies. Several transfusions are necessary, and one is of little value. The authors prefer whole rather than citrated blood.

SANFORD, Chicago.

HYDATID CYSTS OF THE LUNG. J. BONABA and J. A. SOIO, Fac. de med. de Montevideo, 1938, monograph 2.

The roentgenologic diagnosis of hydatid cyst of the lung is further complicated by the addition of the perivesicular pneumocyst. This is characterized by a continuous or discontinuous lineal halo, which is set in motion with changes of position. It may occasionally present a crescent image; with motion this is deformed into a flattened vesicle. During periods of regression it may diminish so in volume as almost to disappear. This is an excellent article and should be read in its entirety by any one interested in the subject. Forty-nine roentgenograms illustrate the article.

SANFORD, Chicago.

ERRONEOUS DIAGNOSIS OF BULLOUS EMPHYSEMA OF THE CHILD'S LUNG. W. MATHEJA, Ztschr. f. Kinderh. **60**:236 (Oct. 15) 1938.

Bullous emphysema is apparently more common in children than in adults, presumably because of the paucity of elastic tissue in their lungs. It is especially common in the presence of delayed resolution during the first two years of life.

Roentgenologically one sees rounded air-containing areas, varying in size from that of a cherry to that of a hen's egg. Cavities may be simulated, but a delicate ring shadow favors the diagnosis of emphysema. If the ring shadow is obscured by consolidated lung tissue, the diagnosis is more difficult. The total clinical picture is important in diagnosis.

Three case reports and roentgenograms accompany the article.

SIEMSEN, Chicago.

Diseases of the Gastrointestinal Tract, Liver and Peritoneum

PERITONITIS DUE TO RUPTURED ACUTE APPENDICITIS IN CHILDREN: INFLUENCE OF DELAY ON THE OPERATIVE MORTALITY. R. ELMAN, Am. J. Digest. Dis. **5**:804 (Feb.) 1939.

This is a study of 181 children operated on for peritonitis at the St. Louis Children's Hospital. In every case the peritonitis followed rupture of an acutely inflamed appendix.

The mortality in the series was 15.5 per cent. One hundred and thirty-eight patients were operated on within eight hours after admission, with a mortality of 14.5 per cent, while 43 were operated on eight to twenty-four hours after admission, with a mortality of 18.5 per cent. This difference is too small to assume much significance from a statistical point of view. These patients were further divided into "toxic" and "nontoxic" groups. The toxic group (33 children) showed a mortality of 70 per cent among those operated on in the first eight hours after admission to the hospital and a mortality of 30 per cent in those in whom operation was postponed nine to forty-eight hours. This period of time was utilized in improving the general condition of the patient; dehydration was corrected, distention was relieved, rest was achieved, transfusions were made and other attention was given. In some cases this delay was long enough to allow localization to occur. The toxic group showed a mortality of 57 per cent, 19 of the 33 patients succumbing. Of the 148 nontoxic patients, 115 were operated on within eight hours, with 4 deaths, or a mortality of 3.5 per cent, while, of 33 whose operation was deferred eight hours or longer, 5 died, a mortality of 15 per cent. The nontoxic group showed a mortality of 6 per cent, 9 of the 148 patients succumbing.

This study tends to show that the problem of deferring an operation depends not so much on the presence or the absence or the degree of peritonitis, which may be difficult to estimate accurately at the bedside, but on the general condition of the child, i. e., the operability. In any case the necessity of hospitalizing every child with acute appendicitis is clearly indicated; immediate or delayed operation can be determined best "on the threshold of the operating room."

MORRISON, Savannah, Ga.

PYOGENIC ABSCESS OF THE LIVER IN A NINE YEAR OLD CHILD. H. A. SLESINGER and H. ANTILIES, Arch. Pediat. **56**:169 (March) 1939.

Abscess of the liver in a 9 year old boy is reported. The source of the infection was a furuncle of the forefinger. The chief symptoms were fever, malaise, pain in the right upper quadrant of the abdomen and hepatic enlargement. Recovery followed incision and drainage.

ORR, Buffalo.

VOMITING IN CHILDREN. E. PRILLA, Illinois M. J. **75**:277 (March) 1939.

This is a general discussion of the causes and treatment of vomiting in infants and children.

BARBOUR, Peoria, Ill.

CHRONIC PEPTIC ULCERS IN CHILDREN. H. B. KELLOGG, Northwest Med. **38**: 129 (April) 1939.

Searching the literature, Kellogg found reports of 36 cases. Forty per cent of the children had severe hemorrhage; 25 per cent had perforated ulcers, and a large number had varying degrees of pyloric obstruction.

The symptoms were much less typical than those in adults, or at least the children did not describe them with the same accuracy. There was some seasonal incidence, and the intensity varied greatly. Many complained merely of mild, indefinite epigastric distress. A few complained of severe boring pains one to three hours after meals. In those in whom pyloric obstruction had developed

there had been mild to severe cramplike pains with nausea and vomiting commensurate with the degree of obstruction.

Pallor, loss of appetite, loss of weight and constipation were frequent. Retarded development was sometimes outstanding.

Kellogg states that when possible the treatment should be dietary, with some means being used to control the gastric acidity. Children tolerate gastric and duodenal operations well, as is evident from the fact that in all the reported cases, regardless of the type of procedure used, the patient recovered. Kellogg presents a case in which the ulcer must have been present for at least three years. The culmination was a perforating duodenal ulcer causing almost complete duodenal obstruction.

ROBERTSON, Omaha.

CONGENITAL ATRESIA OF THE ŒSOPHAGUS. J. W. D. BULL, Brit. M. J. **2**:983 (Nov. 12) 1938.

This is a report of a case of congenital atresia of the esophagus with a tracheoesophageal fistula.

ROYSTER, University, Va.

AN EPIDEMIC OF ALIMENTARY INTOXICATION DUE TO AN ORGANISM OF THE PARATYPHOID B GROUP. P. GIRAUD, C. DE LUNA and J. ODDO, Paris méd. **2**: 73 (July 23) 1938.

Thirty-three children in an orphanage suffered from severe diarrhea, vomiting and fever following the eating of a reheated purée of pea soup. Five of the patients had severe nervous manifestations (coma and tetanic contractures). Three of these died. Bacillus paratyphosus B was isolated from the stools and in some instances from the urine. It was thought that the purée had been contaminated between the first and the second heating.

LESLIE Evanston, Ill.

MEDICAL OR SURGICAL THERAPY OF CONGENITAL PYLORIC HYPERTROPHY (PYLOROSPASM)? A. ECKSTEIN, Klin. Wchnschr. **13**:295 (Feb. 24) 1934.

Eckstein reports observations on 202 patients, of whom 92 were treated medically and 110 surgically. The fatality rate in the first group was 18.4 per cent and in the surgically treated group 3.4 per cent. The duration of clinical care averaged 91 days in the medical group and thirty-four days in the group surgically treated.

SILVERMAN, Syracuse, N. Y.

FAMILIAL JUVENILE CIRRHOSIS OF LIVER: A CLINICAL STUDY. S. AKIBA and J. NAGATA, Orient. J. Dis. Infants **23**:121 (Jan.) 1938.

Akiba and Nagata report detailed observations on juvenile hepatic cirrhosis occurring in 2 members of the same family. The patients were the first girl and the first boy in a family of 6 children. Their deaths occurred at the age of 11 and 12 years, respectively. Both children came to the pediatric clinic of Kyoto Imperial University with the complaint of abdominal swelling and edema of the eyelids. The more important clinical and laboratory findings were: facial edema, pains simulating gallstone, fever, the presence of urobilin and urobilinogen in the urine, anemia, leukopenia and hepatosplenomegaly with ascites. Histologically, the most remarkable feature was the marked proliferation of the small bile ducts, frequently associated with lymphocytic accumulation around the ducts. Nagata calls this pathologic picture cirrhosis hepatis cholangioplastica annularis.

KATO, Chicago.

TWO CASES OF CHRONIC INTUSSUSCEPTION IN CHILDREN. W. NEGISHI, Acta pædiat. japon. **44**:703 (May) 1938.

Two cases of chronic intussusception are reported. One patient was a boy and the other a girl, both 13 years old. Both complained of abdominal pain, and the girl, of vomiting. On examination a distinct oval mass was felt in the abdomen,

with little tenderness. Occult blood was found in the stools of both patients, but frank bleeding occurred only on two occasions in the boy and not at all in the girl. On roentgenographic examination with a barium sulfate meal an invaginating mass was clearly visualized in the first case, and on rectal examination the so-called "claw-like picture" of Lehmann was seen in the second case. On laparotomy the invagination was found to be colicocecal in both patients. Recovery was complete.

KATO, Chicago.

EPIDEMIC ICTERUS. S. TANI, Acta pædiat. japon. **44**:833 (June) 1938.

In 11 cases of epidemic (catarrhal) jaundice the prodromal symptoms consisted of gastrointestinal disturbances (nausea, vomiting, anorexia), pain in the epigastrium and slight fever. Within two to five days icterus of mild degree appeared. Liver enlargement was the most constant clinical finding, and usually the patients complained of some pain from pressure in the right upper quadrant of the abdomen. Bile pigments were found in the urine and in most instances a trace of albumin also. Icterus disappeared in one to three weeks, with complete recovery.

KATO, Chicago.

RESIDUAL NITROGEN, TOTAL SERUM PROTEIN AND ALBUMIN-GLOBULIN QUOTIENT IN DYSPEPTIC TOXICOSIS OF INFANTS. T. TAKATSU, Acta pædiat. japon. **44**:943 (June) 1938.

In 14 cases of dyspeptic toxicosis of infants residual nitrogen was increased to between 34.2 and 83.4 mg. per hundred cubic centimeters of blood. The total protein also was increased, varying from 8.11 to 8.35 mg. The albumin-globulin quotient increased to between 47.2 and 54.7 but returned to normal on recovery.

KATO, Chicago.

AGAR MILK IN DYSPEPSIA AND ENTEROCOLITIS. Y. HIRO and M. NIHARA, Acta pædiat. japon. **44**:957 (June) 1938.

Agar milk (either whole or skimmed milk with addition of 1 per cent agar) had a beneficial effect in dyspepsia and enterocolitis. In mild disorders three days and in severe ones six days was required for agar milk to bring about a noticeable change in the nature of the stools.

KATO, Chicago.

FAMILIAL JUVENILE CIRRHOSIS OF THE LIVER. T. J. VAN DER HOEVEN, Maandschr. v. kindergeneesk. **7**:411 (July) 1938.

Van der Hoeven gives a description of two families in which several children were suffering from cirrhosis of the liver. In the first family 2 brothers died. The clinical description of a sister of 5 years is given. In the second family a brother and a sister died, and a clinical description of another brother is given, who later also died.

VAN CREVELD, Amsterdam, Netherlands.

ROENTGEN DIAGNOSIS OF HYPERTROPHIC PYLORIC STENOSIS IN INFANTS. J. H. NAUTA, Nederl. tijdschr. v. geneesk. **82**:5475 (Nov. 12) 1938.

Attention is drawn to a method by which one is enabled to make a rapid and reliable diagnosis of hypertrophic pyloric stenosis. A few difficulties arising with this method are easily avoided. As the diagnosis based on characteristic clinical signs is sometimes found to be faulty, it is advisable to institute a roentgen examination in every case in which there is a suggestion of hypertrophic pyloric stenosis.

VAN CREVELD, Amsterdam, Netherlands.

Nervous Diseases

GLIOMAS OF THE PONS: CLINICAL AND PATHOLOGIC CHARACTERISTICS. B. J. ALPERS and J. C. YASKIN, Arch. Neurol. & Psychiat. **41**:435 (March) 1939.

Eleven cases of pontile glioma were analyzed from the standpoint of diagnosis and pathology. The pontile glioma usually occurs in children; in 3 of 11 cases it occurred in persons 20, 32 and 45 years of age. At the onset the patient usually has diplopia with some headache, followed within a few weeks by paralysis of the cranial nerves and involvement of the pyramidal and cerebellar tracts. If the tumor is confined to the dorsal part of the pons, the signs are limited almost entirely to paralyses of the cranial nerves, which are numerous. Signs of increased intracranial pressure are usually absent. The course is progressive and may simulate encephalitis. Pontile gliomas are rapidly growing, infiltrating and not removable.

BEVERLY, Chicago.

TWO HUNDRED LUMBAR PUNCTURES OF A SINGLE PATIENT. F. VAN DER BOGERT, Arch. Pediat. **55**:768 (Dec.) 1938.

The 200 lumbar punctures were made in a child with hydrocephalus.

ORR, Buffalo.

GRANULOMATOUS ENCEPHALOMYELITIS DUE TO A PROTOZOAN (TOXOPLASMA OR ENCEPHALITOZOON): II. IDENTIFICATION OF A CASE FROM THE LITERATURE. A. WOLF and D. COWEN, Bull. Neurol. Inst. New York **76**:266 (Dec.) 1938.

Wolf and Cowen describe a case of encephalitis due to a protozoan (Toxoplasma or Encephalitozoon) in an infant who died at 1 month of age. This is the first completely described case to be recorded in the literature, although in 4 other reported cases (all in infants) the disease may have been due to the same cause.

BEVERLY, Chicago.

ACUTE LYMPHOCYTIC MENINGITIS. P. M. RODIER, Rev. franç. de pédiat. **13**:173, 1937.

Ten cases of acute lymphocytic meningitis are reported. A benign course and rapid recovery without sequelae characterize this condition. Rodier thinks the condition is caused by a specific virus. The differentiation from certain types of poliomyelitis, abortive forms of encephalitis and from tuberculous and syphilitic meningitis is discussed.

LESLIE, Evanston, Ill.

NEUROHUMORAL NATURE OF EPILEPSY. N. I. PROPPER, Rev. neurol. **70**:332 (Oct.) 1938.

Propper produced convulsions in rabbits and in dogs by injecting an emulsion of brain from an animal killed immediately after a convulsion produced by electrical stimulation. Emulsions of brain from normal animals did not produce convulsions.

Various authors have shown the influence of various chemicals on the function of nerve endings. Propper has studied the physiology of convulsions since 1928. The study reported here represents the final link in his chain of research.

His results indicate that the injection of this toxic brain produces an excitable state. When this state has been produced, convulsions can be initiated by other forms of stimulation which normally do not produce convulsions. Histopathologic changes occur in the nervous system following convulsions which make the organism more capable of producing further convulsions, thus setting up a vicious circle.

DAFFINEE, Boston.

ARACHNOIDITIS OF THE OPTIC CHIASM CURED BY ENCEPHALOGRAPHIC EXAMINATION. H. ROGER, P. GUILLOT and J. E. PAILLAS, Rev. neurol. **70**:534 (Nov.) 1938.

A girl of 11 years presented marked amblyopia with contraction of the visual fields. Her cure followed two injections of air by the lumbar route, after various other treatment had been found ineffective.

DAFFINEE, Boston.

DIFFERENTIAL DIAGNOSIS OF PONTILE TUMORS AND ENCEPHALITIS. S. A. JORIMANN, Jahrb. f. Kinderh. **147**:151 (Aug.); 260 (Sept.) 1936.

Reports of several cases observed by others are reviewed, illustrating the difficulties involved in distinguishing these two conditions. Numerous reports of cases with pathologic observations are given.

LANDAU, Hannibal, Mo.

CONGENITAL PARALYSIS OF THE FACIAL NERVE AND ABDUCENS NERVE. A. BALINT, Jahrb. f. Kinderh. **147**:256 (Sept.) 1936.

Balint presents the case history of a boy of 5 weeks with paralysis of the left side of the face associated with paralysis of the abducens nerve on the left side. There was no family history of similar conditions in either the maternal or the paternal line.

Several theories as to the cause are advanced, including the theory that there is hypoplasia of the involved nuclear centers, bleeding in these nuclear areas or some peculiar fetal process.

The child died at the age of 3 months.

Pathologic examination showed the presence of a fetal process in the region of the left abducens nerve center, with secondary degeneration of the left facial nerve center.

LANDAU, Hannibal, Mo.

VITAMIN C CONTENT OF THE SPINAL FLUID. J. W. CAMERER, Monatschr. f. Kinderh. **77**:240 (Feb.) 1939.

In healthy breast-fed children under 18 months of age the ascorbic acid content of the spinal fluid averages 4.2 mg. per hundred cubic centimeters. It is definitely lower in artificially fed children. Rachitic children do not differ from nonrachitic, artificially fed children, but in patients with spasmophilia the content of ascorbic acid in the spinal fluid drops to an average of 0.85 mg. per hundred cubic centimeters. Minor infections have no influence, but long-standing disease causes diminution. In patients with nutritional disturbance the diminution is in proportion to the duration of the condition. A diet low in ascorbic acid results in marked diminution of this vitamin in the spinal fluid. Taking all artificially fed children, both healthy and sick, one finds the average content of ascorbic acid in the spinal fluid to be 2.13 mg. per hundred cubic centimeters.

During the first two years there is no relation between age and ascorbic acid content of the spinal fluid.

In healthy children with low vitamin C content, the content can be raised easily to 5.5 mg. per hundred cubic centimeters by adding vitamin C to the diet.

GERSTLEY, Chicago.

EFFECT OF ROENTGEN IRRADIATION ON SARCOMA OF THE CEREBELLUM. I. FÉNYES and P. KISS, Orvosi hetil. **82**:688 (July 9) 1938.

The authors report a case in which the diagnosis was tumor in the vermis. The operation did indeed disclose a big tumor of the vermis which seemed to be inoperable. A small part of the tumor was removed, and the wound was closed. Histologic examination showed sarcoma fusocellulare. After the operation roentgen examinations were made, and the child received 2,600 roentgens in treatment in nine

weeks. The irradiations brought a little improvement, but in the end the child died with symptoms of tumor of the spinal cord. Histologic examination of the cerebellum showed that the tumor cells in the cerebellum had disappeared and that their place was occupied by connective tissue. This examination shows that the irradiation really had a great effect on the tumor cells.

GÖTTCHE, Budapest, Hungary.

CATALEPTIC SYMPTOMS IN CHILDHOOD. D. MÓRITZ and G. KÖNIG, Orvosi hetil. **82**:855 (Aug. 27) 1938.

Cataleptic symptoms were observed in 4 children. The patients did not reveal any neurologic alterations, and their mental development was also normal. The previous history did not give any valuable data, so that the authors think that these cataleptic symptoms can occur in childhood after very slight psychic or toxic troubles because children have a "catatonoid reactibility". All these cataleptic symptoms disappeared later.

GÖTTCHE, Budapest, Hungary.

NEUROLYTIC FERMENTS IN CEREBROSPINAL FLUID OF HEALTHY AND SICK CHILDREN. E. KOVÁCS, Orvosi hetil. **82**:928 (Sept. 17) 1938.

Kovács tried to show neurolytic ferments in cerebrospinal fluid from various groups and found that the cerebrospinal fluid of healthy children has no neurolytic effect. That of children with meningitis has very slight neurolytic effect. The greatest such effect was found in spinal fluid from patients with epilepsy.

GÖTTCHE, Budapest, Hungary.

DIFFERENTIATION BETWEEN MENINGITIS AND ENCEPHALITIS BY THE COLOR REACTION OF THE CEREBROSPINAL FLUID. P. UJSÁGHY, Orvosi hetil. **82**:1068 (Oct. 29) 1938.

In the presence of alphanaphthol carbohydrates give with sulfuric acid a bluish red color. If there is lactic acid in the cerebrospinal fluid, the reaction becomes yellow. In cases of meningitis one gets a yellow reaction because more or less lactic acid is always present, whereas in cases of encephalitis one gets a bluish red color. The lactic acid has to reach a quantity of 25 to 30 mg. per hundred cubic centimeters of cerebrospinal fluid before the yellow reaction occurs.

GÖTTCHE, Budapest, Hungary.

CEREBRAL DEFECT IN CHILDREN BORN BY CESAREAN SECTION. T. BRANDER, Acta pædiat. **23**:145, 1938.

The opinion is widely held that cesarean section provides insurance against the possibility of cerebral injury to the fetus; the infant mortality in the larger series of reported cases approximates 0 per cent. That this optimism is not well grounded is attested by the fact that Brander was able to find recorded in the literature 72 cases of anatomically proved intracranial injury of the infant delivered by laparotomy. Cesarean section is attended by the following sources of danger to the fetus:

(*a*) Before section: unsuccessful attempts to apply forceps; unduly prolonged or unusually severe labor; artificial engagement of the head by use of Walcher's position; contracted maternal pelvis.

(*b*) In association with operation: vaginal section; removal of the fetus by the feet; instrumental delivery of the head; insufficiently long uterine incision; accidental incision of the placenta.

These possibilities for injury are the more apt to be realized if the infant is premature or if a hemorrhagic or toxic state coexists. Schultze's method of resuscitation is hazardous. Three illustrative cases are reported for the first time, and an extensive bibliography is supplied.

MCCUNE, New York.

Psychology and Psychiatry

STUDIES IN MONGOLISM: II. THE THYROID GLAND. C. E. BENDA, Arch. Neurol. & Psychiat. **41**:243 (Feb.) 1939.

Benda studied the thyroid glands of 14 children with mongolism, most of whom died within the first year of life, and compared them with the glands of normal children of the same age. The pathologic condition of the glands indicates that "hyperthyroidism during the prenatal period is a cornerstone on which rests the explanation of mongolism."

The pathologic picture of the thyroid glands of patients with mongolism suggests the histologic changes found in the thyroid glands of patients with pituitary disease.

BEVERLY, Chicago.

CLINICAL MONGOLISM IN THE NEGRO. R. B. SCOTT, Arch. Pediat. **56**:4 (Jan.) 1939.

Mongolism is reported in 6 Negro children. Four of the children were girls and 2 were boys.

ORR, Buffalo.

DYSPHONIA ASPECTS OF MONGOLIAN IMBECILITY. C. H. VOELKER, Arch. Pediat. **56**:114 (Feb.) 1939.

There are four aspects to the dyslogia of children with mongolism: (1) the diplocic aspect, which is improved by attention to duration of intonation and loudness of intonation; (2) the dysphonoid aspect, which is improved by emphasis again on duration of intonation; (3) the dyslaloid aspect, which is improved by adjustments in articulation, and (4) the dysphonic aspects, which are improved by dramatization of the inflections.

There are two aspects to the dysphonoid characteristics of the child with mongolism: tracheophonia and pneumophonia. The cardinal phoniatric characteristics of pneumophonia are roughness, breathiness, huskiness or hoarseness of voice; those of tracheophonia are thickness and gutturality of voice. In the former there are two aspects of the voice disorder demanding medical attention, iatrogenic tracheophonia and rhinophonia clausa.

Mogiphonia is relieved when the patient learns to retract the protruding tongue.

Gutturophonia is removed in the very dramatization of the speech situation, employing as it does what the musicians call voice placement.

Pneumophonia and ventriculophonia are removed by training in pitch of intonation, a training regularly employed by musicians, although rarely used by phoniatricians, in other voice disorders of the same type.

For general purposes the vocal difficulties of the child with mongolism may be divided into two parts: (1) the glossal, resulting in thickness and gutturality, and (2) the laryngeal, resulting in hoarseness and roughness.

ORR, Buffalo.

ETIOLOGIC STUDY OF MONGOLISM. R. TURPIN, Union méd. du Canada **67**:1263 (Dec.) 1938.

Turpin, after a study of 104 cases of mongolism, concludes that the most important factor in the production of this condition in a child is the age of the mother. If procreation were limited to women between 20 and 30 years of age, the frequency of mongolism would be reduced at least two thirds. A comparison of familial statistics showed many more psychotic persons among the parents of children with mongolism. There were more stillbirths in these families, and the infant mortality was higher. Dystrophies of all sorts were more common in these families. In fact, Turpin asserts that one third of the siblings of his patients presented one or another anomaly.

DAFFINEE, Boston.

EMOTIONAL AND NERVOUS DISEASES IN PEDIATRICS FROM THE CLINICAL AND SOCIAL POINTS OF VIEW. TOPIC I. THE FOURTH INTERNATIONAL PEDIATRIC CONGRESS, ROME, SEPTEMBER 1937. Acta pædiat. **22**:1, 1938.

Fourteen papers summarize contemporary opinions relating to the broad topic announced in the title. The truly international character of the symposium is brought out by the fact that more than a dozen countries were represented either in the principal presentations or in the discussion thereof. The heaviest emphasis is placed on the prevention of reactive emotional disorders in children through the medium of education.

McCUNE, New York.

DOES THE BIRTH OF MENTALLY DEFECTIVE CHILDREN SHOW A SEASONAL FLUCTUATION? T. BRANDER, Acta pædiat. **23**:165, 1938.

In the attempt to get at the whole truth of the pathogenesis of mental defect, recourse has often been made to certain types of peristatic causes referred to in general as seasonal influences; the seasonal consumption of alcohol around important holidays may be taken as an example of what is meant by this classification. Statistics have been published that pretend to prove the significance of these seasonal influences inasmuch as they demonstrate periodic annual fluctuations in the birth frequency of defective children. Brander points out that the majority of these statistics are rendered invalid by lack of control data relating to the birth rate of the normal members of the population. He illustrates his point and proves his argument as follows: Of 1,791 mentally defective children whose records were reviewed, the majority were born between June and August and the smallest number between December and February; however, the seasonal birth frequency of 1,485 normal children was precisely the same as that of the defectives. The bibliography compiles about 50 references to an interesting aspect of biometry.

McCUNE, New York.

Diseases of the Genitourinary Tract

RHABDOMYOSARCOMA OF THE URINARY BLADDER. E. UHLMANN, A. GROSSMAN and J. K. CALVIN, Ann. Surg. **109**:624 (April) 1939.

Rhabdomyosarcoma arising from the musculature of the bladder is rarely seen but of special interest because of the grave prognosis and the difficulty of treating for it. Seven cases have been reported in the literature.

The growth frequently occurs in the form of grapelike masses, suggesting hydatidiform mole. Usually the tumor is of a white to grayish yellow color and has a fleshy consistency. The surface is usually smooth and translucent.

The symptoms associated with this tumor are variable. Occasionally there are no genitourinary symptoms, the only complaint being abdominal pain. Hematuria is rare, particularly early. Owing to the infiltration of the neck of the bladder, retention of urine is a frequent occurrence. The diagnosis is frequently made accidentally during exploratory operation.

The prognosis is grave. None of the patients whose cases have been reported survived more than one year after operation.

The authors report in detail their observations on this condition in a 10 month old boy.

BANTIN, Omaha.

ECLAMPSIA-LIKE SYNDROME OCCURRING IN PREGNANT DOGS AND RABBITS FOLLOWING RENAL ARTERY CONSTRICTION. L. V. DILL and C. C. ERICKSON, Proc. Soc. Exper. Biol. & Med. **39**:362 (Nov.) 1938.

In 8 pregnant mongrel dogs the renal arteries were clamped so as to produce minimal to moderate constriction. Within forty-eight to one hundred and twenty hours the dogs showed weakness, lassitude, coma and convulsions; they also exhibited hypertension, hematuria, albuminuria and retention of nitrogen. One

animal delivered her young, with subsequent improvement, and is well after eight months of sustained hypertension. Another delivered 5 stillborn and macerated fetuses, after which she showed rapid improvement. Five animals died. Two were put to death.

The renal arteries were constricted in 5 nonpregnant females and 2 control males. One male is living after eleven months and one female after six months with marked hypertension. In none of the controls did the rapidly fatal condition occur.

In the 7 pregnant dogs at autopsy there were evidences of acute cardiac failure with pulmonary edema and myocardial changes. The kidney and liver presented changes similar to those characterizing human eclampsia.

A similar picture was produced in another experiment with rabbits.

These preliminary observations indicate that renal ischemia or renal injury experimentally produced in pregnant dogs and rabbits results in an eclampsia-like syndrome characterized by a rapidly fatal course and significant pathologic lesions in the liver and kidneys.

HANSEN, Minneapolis.

NEPHROPATHIES IN THE COURSE OF ACUTE NASOPHARYNGEAL DISEASES OF INFANTS. D. TIMUS and V. PETRESCU-COMAN, Med. inf., Roumania **1**:104 (Feb.) 1939.

Timus and Petrescu-Coman show that aside from the usual diseases which determine renal lesions in a baby, severe transient infections of the rhinopharynx are important. In methodic examinations of the urines of babies having coryza, pharyngitis or tonsillitis they have noticed that in almost all there are renal lesions varying as to intensity and duration. The development of nephritis is favored by many factors: a certain renal predisposition, congenital syphilis, lymphatism and status thymicolymphaticus. To these the authors add from their own observation an advanced state of dystrophy. The renal lesions are manifested by edema, vomiting and diarrhea. Examination of the urine shows albumin, red cells, leukocytes and in severe cases granular casts and desquamated epithelium of the urinary tract. The authors record a series of cases in which renal lesions of variable intensity and course were shown, among others follicular tonsillitis, pharyngitis and otitis, and repeated tonsillitis. In cases in which after recovery from nasopharyngeal lesions there persists an unexplained subfebrile state, it is necessary to examine the urine, which in the majority of cases will indicate the presence of renal lesions.

FROM AUTHORS' SUMMARY.

RUPTURE OF KIDNEY. H. KUNZE, Beitr. z. klin. Chir. **165**:105, 1937.

Here is a fairly complete discussion of problems arising from rupture of the kidney.

The author emphasizes the fact that rupture may be caused by intrinsic as well as extrinsic factors. The prognosis in most cases is excellent. Cicatrization occurs in about ten days on the average.

Six patients were examined one year after injury, and in none was traumatic nephritis found. Six of 12 patients had urobilinogen in the urine, and 2, albumin.

The management of these injuries varies—it depends entirely on the individual case. If possible, conservative treatment and watchful waiting should be practiced. Early operation is advised if there is marked acute anemia. The quick healing and good prognosis of the ordinary injury of the kidney give one the opportunity to trust nature and give human healing power a chance.

RUBIN, Boston.

SURGICAL MANAGEMENT OF EXTROPHY OF THE BLADDER. K. LUHMANN, Beitr. z. klin. Chir. **165**:221, 1937.

Luhmann goes into detail about the surgical management of extrophy of the bladder. He cites the experience of others as well.

RUBIN, Boston.

TREATMENT OF BACILLUS COLI INFECTION OF THE URINARY TRACT WITH MANDELIC ACID. H. KUNSTMANN, München. med. Wchnschr. **85**:790 (May 27) 1938.

Kunstmann discusses the experience of American and English investigators with compounds of mandelic acid in urinary infections. In his own series of 16 patients with pyelitis, pyelonephritis and cystitis due to Bacillus coli there were 13 cures. Symptomatic improvement and clearing of the urine occurred in all in from three to eight days. In only 2 patients was the urine not sterilized. Kunstmann found that ammonium mandelate was tolerated better than the calcium salt. Mandelic acid treatment is harmless and is effective without local treatment.

BRAHDY, Mount Vernon, N. Y.

DIAGNOSIS OF HEMATOCOLPOS. C. HIRSCHBERGER, Acta pædiat. **23**:229, 1938.

Although the possibility of hematocolpos is doubtless known to every pediatrician, it is apt to occupy a position far back in his mental storehouse; for this reason a patient presenting the condition is likely to be sent to a surgeon with the erroneous diagnosis of abdominal tumor. Hirschberger reports a classic case, in which a 14 year old girl who had never menstruated complained suddenly of urgency of urination, pain in the lower abdominal region and constipation. A tumor in the lower part of the abdomen was evident from inspection and from palpation. Catheterization failed to produce diminution in the size of the mass. The midline position of the tumor was considered to be against the diagnosis of ovarian tumor, either cystadenoma or dermoid. Pregnancy was excluded. The diagnosis of hematocolpos or hydrocolpos was made correctly when an imperforate hymen was detected. Operation with the patient under general anesthesia resulted in the liberation of a large volume of blood-stained fluid and produced disappearance of the "tumor." Reports of about 40 cases of this kind have been published. Although distention of the vagina by the secretions retained behind an imperforate hymen may occur in an infant, the secretory activity of the immature vaginal epithelium is so small that such an occurrence is not usually encountered before the onset of puberty.

MCCUNE, New York.

Diseases of the Ductless Glands; Endocrinology

THE INFANT HERCULES TYPE OF ADRENO-GENITAL SYNDROME. L. DODS and R. JEREMY, M. J. Australia **1**:1047 (June 18) 1938.

Dods and Jeremy describe a condition in a 12 year old boy which they feel falls into the category of adrenal cortical disturbances. The literature is briefly reviewed, and a method of diagnosing adrenal lesions by the use of perirenal insufflation of air and roentgenography is described. The roentgen and laboratory findings are given in detail.

GONCE, Madison, Wis.

LIPODYSTROPHY AND LIPOMATOSIS FROM REPEATED INJECTIONS OF INSULIN. P. NOBÉCOURT and P. DUCAS, Arch. de méd. d. enf. **42**:193 (April) 1939.

A girl of 16 years in whom diabetes had developed at the age of 9 and who had received several injections of insulin daily either in the thigh or in the hip for a period of six years had at these sites, at the time of examination, small tumors with all the physical aspects of lipomas. These tumors varied in size from that of a small nut to that of an orange and were painless and elastic. The skin was streaked and bluish.

With the introduction of protamine zinc insulin, one injection daily in the arm, the tumors of the thighs rapidly disappeared, but those on the hips regressed slowly and could readily be seen a year later. Estimations of the blood cholesterol were within normal limits.

A girl of 14 who had suffered from diabetes for six years showed, after several years of daily injections of insulin in the thigh, atrophic areas as large as the palm of the hand and about 2 cm. in depth. The skin was normal in appearance but could not be picked up easily and seemed to be deprived of all subcutaneous fat. These atrophic areas remained unchanged after a year, although protamine zinc insulin was substituted and different sites of injection insisted on.

A third observation, similar to the latter, is reported concerning a girl of 18 who had been diabetic since the age of 10.

A review of the now abundant literature on this condition, the pathologic nature of which is not understood, terminates the contribution.

AMESSE, Denver.

EFFECT OF PREPUBERAL CASTRATION OF THE FEMALE ALBINO RAT ON THE THYMUS AND THE LYMPH GLANDS. H. CHIODI, Rev. Soc. argent. de biol. **14**:222 (June) 1938.

After investigating female albino rats which were castrated prepuberally (at 30 days of age) and observed until they were 200 days of age, Chiodi concludes that the thymus of the castrated female is heavier than that of the normal control of equal age level. The increase in weight is due to thymic tissue. The weight curve of the thymus in the castrated female assumed a form similar to that of the control animal but reached a higher level. The time of onset of physiologic involution of the thymus was not affected in either sex by prepuberal castration. A slight increase in the weight of submaxillary, cervical and axillary lymph nodes was observed, and the time of physiologic involution of these nodes differed from that of the thymus.

KALISKI, San Antonio, Texas.

EFFECT OF THYROIDECTOMY AND OF ADRENALECTOMY ON THE THYMUS OF THE CASTRATED WHITE RAT. H. CHIODI, Rev. Soc. argent. de biol. **14**:322 (July) 1938.

After inducing thymic hypertrophy in albino rats of both sexes by prepuberal castration, Chiodi subjected the rats to thyroidectomy and observed that this caused a reduction in thymic size. He concludes, therefore, that the presence of the thyroid is necessary to maintain the thymic hypertrophy which follows prepuberal castration. Adrenalectomy in castrated females, on the other hand, increased slightly the thymic enlargement.

KALISKI, San Antonio, Texas.

EFFECT OF INJECTIONS OF EXTRACT OF THYMUS (HANSON) ON THE GROWTH OF THE ALBINO RAT. H. CHIODI, Rev. Soc. argent. de biol. **14**:326 (Aug.) 1938.

Chiodi prepared an extract of the thymus gland of the young lamb by removing the extracapsular connective tissue and grinding the parenchyma. Mixing this with 0.5 per cent hydrochloric acid and keeping the mixture in a water bath for one and a half hours at a temperature oscillating between 92 and 96 C., he obtained after filtration a clear yellow liquid with the odor of soup, containing 0.370 Gm. of albumin per hundred cubic centimeters as determined by the micro-Kjeldahl method. This extract injected into five successive generations of albino rats did not produce any acceleration of growth. Chiodi expresses surprise that his results were completely negative and thus failed to confirm those of Rowntree.

KALISKI, San Antonio, Texas.

EFFECTS OF THYMECTOMY ON THE GROWTH OF ALBINO RATS. H. CHIODI, Rev. Soc. argent. de biol. **14**:383 (Sept.) 1938.

Employing albino rats, Chiodi found that thymectomy did not result in any alteration of somatic growth or of the weight of the lungs, heart, liver, kidneys, gonads or glands of internal secretion. Histologic examination of the bones did not reveal any differences between the thymectomized animals and the controls. Because

of the limited number of animals used (28 thymectomized males and 24 controls, 10 thymectomized females and 14 controls), the author hesitates to make categoric affirmation of his conclusions.

KALISKI, San Antonio, Texas.

EFFECT OF CASTRATION ON THE WEIGHT OF THE THYMUS IN THE ADULT ALBINO RAT. H. CHIODI, Rev. Soc. argent. de biol. **14**:435 (Nov.) 1938.

Chiodi reports that castration of the adult albino rat induces hypertrophy of the thymus. This hypertrophy was demonstrated thirty, forty-five and sixty days after castration. Histologically he observed a tendency for lymphocytes to disappear from the medullary zone and the occurrence of interstitial hemorrhages in the thymic tissue of the castrated animals.

KALISKI, San Antonio, Texas.

HYPERINSULINISM. J. GELDRICH, Orvosi hetil. **82**:701 (July 16) 1938.

A 2 year old boy had hypoglycemic attacks after light influenza. These attacks reappeared several times, and in the end purulent meningitis developed, and the child died. The autopsy showed an increase in the number and size of the islands of Langerhans. The values were normal for blood sugar under normal conditions; hypoglycemia occurred only in attacks after starvation or after banal infections of the upper respiratory tract. These attacks naturally diminished with increased resistance of the organism against infections.

GÖTTCHE, Budapest, Hungary.

LOCAL LIPOMATOSIS CAUSED BY INSULIN INJECTIONS. B. WOLLEK, Orvosi hetil. **82**:904 (Sept. 10) 1938.

A 4½ year old child had painless springy swellings on both arms and both thighs at the places where injections of insulin had been given. These swellings corresponded perfectly to those described by Priesel as "local lipomatosis." The alterations began to develop after a course of insulin treatment of ten months and reached their present size in two years. During two and one-half years the child received daily 20 units of insulin in three injections.

GÖTTCHE, Budapest, Hungary.

Diseases of the Muscles, Bones, Joints and Lymph Glands

LACTIC ACID FORMATION BY MUSCLES OF SCORBUTIC GUINEA PIGS. O. RAHN, Proc. Soc. Exper. Biol. & Med. **39**:334 (Nov.) 1938.

The excised muscle of scorbutic guinea pigs produces only half as much lactic acid as the muscle of normal animals. The injury of the enzyme complex produced by scurvy is not repaired by the simple addition of ascorbic acid to the minced muscle; i. e., 100 mg. of ascorbic acid added to 1 Gm. of muscle has no effect on the final amount of lactic acid produced either with normal or with scorbutic muscle.

HANSEN, Minneapolis.

CHEMICAL COMPOSITION OF TRANSVERSE METAPHYSEAL BANDS PRODUCED IN GROWING BONES BY PHOSPHORUS INGESTION. C. O. ADAMS, Proc. Soc. Exper. Biol. & Med. **39**:351 (Nov.) 1938.

Twenty-nine young rabbits were given 0.6 mg. of yellow phosphorus daily in addition to the stock diet. Phosphorus lines were demonstrated by roentgen examination during the second week. Three of these animals and three normal litter mates as controls were selected for a study of the mineral composition of the bones. A section of the transverse zone of increased density of the metaphysis was excised from each test rabbit and a similar section was taken from each control. A section of the cortex was taken from each group.

The percentages of calcium and phosphorus and the calcium-phosphorus ratio were the same in the phosphorus-treated bone as in the control bone. The increase in density represents deposition of radiopaque salts in greater amount per unit of tissue calcified, since calcification and ossification have continued unchecked while the cartilaginous growth has been inhibited. This suggests that the zones of increased density in metaphyses of growing bones produced by ingestion of phosphorus are in fact "growth arrest lines."

HANSEN, Minneapolis.

CRANIOTABES. L. THATCHER, Tr. Med.-Chir. Soc. Edinburgh, 1938, p. 181; in Edinburgh M. J., October 1938.

Two infants about 4 months of age who had been fed inadequately on cow's milk since the early days of life showed poor nutrition with patchy softness of the bones of the head. Particularly did the occipital bone show decalcified parchment-like areas. These children gave no evidence of rickets, spasmophilia or congenital syphilis, but there had been conditions which interfered with feeding and the general nutrition, and these evidently caused interference with the metabolism of calcium and phosphorus so that calcium was removed from the bone. Under normal feeding, including cod liver oil, the bones became normal along with improvement in the health of the children.

NEFF, Kansas City, Mo.

ROENTGENOGRAPHIC APPEARANCE OF THE NOTCHES IN THE CHILD'S VERTEBRAL BODIES. P. PASSEBOIS and P. BÉTOULIÈRES, Presse méd. **47**:280 (Feb. 18) 1939.

A notching of the anterior borders of the vertebral bodies appears in the dorsal vertebrae of the infant of 4 or 5 months. The maximum development is at 15 to 20 months. The lesions disappear entirely during adolescence. Those in the fifth, sixth and seventh dorsal vertebrae are the last to disappear, being present up to the age of 16 or 17 years. The classic explanation of this phenomenon is that they represent a vascular nutritive canal. The authors discount this view on the basis of histologic studies, considering the notches due to the presence of small globules of medullary tissue within the vertebral bodies. They hazard the guess that these formations may be hemopoietic in function.

DAFFINEE, Boston.

TREATMENT OF ACUTE OSTEOMYELITIS IN ADOLESCENTS. P. PETIT, Presse méd. **47**:284 (Feb. 22) 1939.

Recently the treatment of osteomyelitis has been revised. Trephining is considered a blind and brutal method that often spreads the infection to the marrow cavity. By subperiosteal resection, however, the entire focus of infections can be removed. Regeneration of bone then completes the cure.

DAFFINEE, Boston.

HEREDITARY MUSCULAR ATROPHY (LEYDEN-MOEBIUS TYPE) TREATED WITH VARIOUS AMINO ACIDS. L. BÉTHOUX, GAUTIER and J. ROCHEDIX, Rev. neurol. **70**:629 (Dec.) 1938.

A 10 year old boy presented the typical picture of diffuse atrophy of the trunk muscles. Symptoms had appeared a year previously. He was given histidine, tryptophan, aminoacetic acid and cystine by injection and aminoacetic acid by mouth. A remarkable clinical improvement occurred, and at the end of five months he had gained weight, grown taller and was able to climb stairs with less effort. His creatinine-creatine ratio improved in a month of treatment. Weekly determinations were 0.105, 0.51, 1.22, 1.73 and 0.934. He was taken home at the insistence of his parents and without treatment showed an extension of the atrophy a year later.

DAFFINEE, Boston.

INTERSTITIAL MYOSITIS FIBROSA IN A GIRL SEVEN YEARS OF AGE. L. KAGAN, Jahrb. f. Kinderh. **147**:207 (Aug.) 1936.

The parents brought a 7 year old girl to the hospital with hypertrophy of the tonsils and marked dental caries. The illness began with a slight elevation of temperature and pain in the face, upper extremities, back and chest. After a short time the upper extremities were bent at the elbows, the neck was bent forward, and there was inability to open the mouth wide. The jaw muscles and the muscles of the upper part of the arms were quite hard. There was slight pain on light pressure but no spontaneous pain.

Injections of milk and a substance containing albumin, lipoid and fat, hot baths, massage and exercise were used therapeutically. These measures were followed by recovery.

Despite lack of histologic evidence the diagnosis of myositis fibrosa interstitialis was made because of the more or less typical and favorable course.

LANDAU, Hannibal, Mo.

CLINICAL ASPECTS OF MYATONIA CONGENITA. E. GLANZMANN and D. OKONO, Jahrb. f. Kinderh. **147**:241 (Sept.) 1936.

The symptoms and objective evidences of myatonia congenita are described. Several case reports are given. The possibility of a relationship with cases of nuclear aplasia is discussed because of the presence of similar associated abnormalities in many instances of both types of conditions.

A case history of a combination of the two in a child of 2 years, with positive evidence of both nuclear aplasia and myatonia congenita, is given.

LANDAU, Hannibal, Mo.

A REVIEW OF THE DIFFERENTIAL DIAGNOSIS OF THE OSTEOPATHIES DUE TO HORMONAL CAUSES, AVITAMINOSIS AND KIDNEY DISEASE. H. R. SCHINTZ, Röntgenpraxis **11**:1 (Jan.) 1939.

This is a 14 page didactic lecture which supplies a classification of the diseases of the skeleton due to or associated with causes of the categories mentioned in the title. The features diagnostic of each of the entities are presented with admirable clarity in tabular form. Although the text is in German, the summaries of diagnostic points are couched in terms that are largely of Latin origin; hence the paper will be found useful to those who disavow familiarity with any language other than English.

McCUNE, New York.

ATONIC-SCLEROTIC MUSCULAR DYSTROPHY (TYPE ULRICH). E. STOEBER, Ztschr. f. Kinderh. **60**:279 (Oct. 15) 1938.

Ulrich first described the condition in 1932. In his 2 cases the process was congenital and characterized by marked sclerotic changes in the skeletal musculature except that of the distal portions of the extremities, which was flaccid, and by normal findings in regard to the nervous system.

Stoeber describes in detail a case which was similar in most respects to those described by Ulrich. A more or less fixed fetal habitus was characteristic. There was profuse sweating. The mentality seemed normal.

SIEMSEN, Chicago.

Skin Diseases; Allergy

PROPHYLAXIS IN ALLERGY. R. A. KERN, Ann. Int. Med. **12**:1175 (Feb.) 1939.

After a discussion of the known facts relative to the production of sensitivity in those so predisposed, Kern offers as a means of preventing sensitization of the fetus in utero the avoidance of overindulgence in foods by the pregnant mother.

Prevention of sensitization to inhalants is accomplished by attention to the sleeping quarters, choice of toys, pets and furniture.

The importance of selecting a suitable occupation is urged on the allergic person.

Special care should be exercised in the selection of places for vacations with reference to plant pollination.

It is suggested that attempts at prophylaxis against hypersensitiveness should be made in a child with an allergic parent, in any person who has had allergic manifestations in the past, in a person who at present has some minor allergic symptom and in one with obvious allergic disease.

It is estimated that 15 per cent of the white population of this country is allergic.

READING, Galveston, Texas.

THE TRICHOPHYTIN TEST: ITS VALUE AS A DIAGNOSTIC AID. G. M. LEWIS, G. M. MACKEE and M. E. HOPPER, Arch. Dermat. & Syph. **38**:713 (Nov.) 1938.

The trichophytin test was studied on over 1,000 patients during a period of three years. Patients who received the test dose of trichophytin were examined for evidence of a fungus disease, and when foci were found, both microscopic tests and cultural studies were undertaken.

The results show that a positive reaction to trichophytin is not in itself diagnostic; when such a reaction agrees with other findings, confirmation is obtained, the prognosis is hopeful, and treatment should be conservative. If the reaction is negative in the presence of an eruption proved to be due to a fungus, the immediate prognosis is poor, and treatment should be vigorous and sustained. If the reaction is negative when examination for a fungus reveals no fungus and the primary eruption is inflammatory, one is justified in calling the rash non-mycotic.

JACKSON, Iowa City.

CONGENITAL ECTODERMOSES IN INFANCY AND CHILDHOOD. B. L. KAHN, Arch. Pediat. **56**:150 (March) 1939.

Von Recklinghausen's disease in a girl aged 7 is reported. Endocrinologic defects (hypothyroidism), neuropsychiatric abnormalities and cutaneous manifestations were demonstrable. The relationship of von Recklinghausen's disease to other ectodermic abnormalities is discussed.

ORR, Buffalo.

TREATMENT OF ANTISERUM TO REMOVE SERUM-SICKNESS-CAUSING FACTOR. L. R. JONES and M. S. FLEISHER, J. Bact. **36**:300 (Sept.) 1938.

For years Jones and Fleisher have been doing interesting work in connection with the production of serum sickness in animals. They subjected phenolized and nonphenolized portions of a lot of tetanus antitoxin (whole horse serum) to various modifications of a method for the removal of the factor causing serum sickness in the rabbit. The method involved addition of dilute alkali to the serum, application of heat at 50 or 55 C. to this mixture for one or one and one-half hours and subsequent neutralization with an equivalent amount of acid. After restoring the diluted serum to its original volume by evaporation, it was tested in rabbits for its activity in causing serum sickness and titrated for antitoxic content by protection tests in guinea pigs against known doses of toxin. The effect of varying the concentration of alkali, the temperature and the period of application on the factor causing serum sickness and on the antitoxic activity is described. In dealing with the sample of nonphenolized serum it was found possible to adjust the method to the particular requirements of the lot of serum under examination in such a way as to remove all of the factor causing serum sickness and yet retain 80 per cent of the original antitoxic content. Phenolized serum required somewhat more drastic treatment for the removal of the factor causing serum sickness. There was a somewhat greater destruction of antitoxin.

STOESSER, Minneapolis.

RAT ACRODYNIA AND THE ESSENTIAL FATTY ACIDS. F. W. QUACKENBUSH, B. R. PLATZ and H. STEENBOCK, J. Nutrition **17**:115 (Feb.) 1939.

Acute cutaneous lesions developed on rats in four or five weeks when these rats were given a basal diet of purified casein dextrose and salts supplemented with carotene, synthetic B_1 and riboflavin. Chronic lesions ("rat acrodynia") appeared if the animals were given fat supplements at subcurative levels. Peanut oil or wheat germ oil completely cured either type of dermatitis. The fatty acid fraction of peanut oil and the ethyl esters prepared from the saponifiable fraction of wheat germ oil showed potency.

FREDEEN, Kansas City, Mo.

DIAGNOSIS OF INFANTILE ECZEMA. P. WORINGER, Rev. franç. de pédiat. **13**:117, 1937.

An excellent description of the lesions and course of infantile eczema is given with a discussion of points which differentiate it from seborrheic eczema and other similar dermatoses. Woringer is convinced of the allergic nature of the condition and that it is inherited as a recessive characteristic. In addition to skin tests to determine the offending article, he uses the method of Prausnitz and Küstner of injecting serum obtained from the infant into a nonallergic person and making a cutaneous test on that person.

LESLIE, Evanston, Ill.

A CASE OF XANTHOMA MOLLUSCIFORME ET GENERALISATUM IN INFANCY. D. STEIGER-KAZAL, Orvosi hetil. **82**:758 (July 30) 1938.

In infants xanthoma is rare, but it may sometimes be inborn, as in the present case. At birth the 19 month old infant had some little knots on the skin. In the sixteenth month they increased suddenly. Histologic examination verified the diagnosis. These xanthomatic nodules were very small, but their number was very great; even the skin of the hairy scalp was full of them. The cholesterol content of the blood was normal. The author tried treatment with liver extract, but without success.

GÖTTCHE, Budapest, Hungary.

Miscellaneous

SOME LIMITATIONS IN PREVENTIVE MEDICINE. H. A. CHRISTIAN, Ann. Int. Med. **12**:1499 (March) 1939.

The effectiveness of preventive measures on the communicable diseases is clearly shown. As preventive measures succeed there occurs an increase in the nonpreventable diseases. The increase in life expectancy has increased the number of older persons, but the "life span" has not been increased. There are a greater number of older persons below par who are subject to degenerative diseases which are not preventable. Such changes are increasing the importance of geriatics at the expense of pediatrics.

The nearly complete elimination of communicable or contagious diseases may create a soil from which will arise extensive epidemics of these diseases with a high mortality.

The need for physicians will increase, but they will deal more and more with older and less and less with younger patients.

READING, Galveston, Texas.

A DELAYED TOXIC REACTION TO SULPHANILAMIDE. L. DANZIGER, Bull. Johns Hopkins Hosp. **63**:340 (Nov.) 1938.

A 19 year old youth with gonorrheal urethritis showed cyanosis, confusion and negativism after four days' treatment with sulfanilamide. One month later a second course of treatment produced the same symptoms. There were no residuals.

LYTTLE, New York.

STUDIES IN THE PHYSIOLOGY OF ARTIFICIAL FEVER: I. CHANGES IN THE BLOOD VOLUME AND WATER BALANCE. J. G. GIBSON and I. KOPP, J. Clin. Investigation **17**:219 (May) 1938.

Artificial fever produced by physical means is characterized by a large loss of water from the blood stream by way of the skin and lungs. While patients vary in their response to artificially induced high temperatures, there exists for each a blood volume beyond which further reduction in volume leads to peripheral vascular collapse. The prevention of shock in artificial fever therapy is dependent on giving fluids in amounts and by routes adequate for the maintenance of the blood volume and water balance.

GUEST, Cincinnati.

A STUDY OF SOME OF THE PHYSIOLOGICAL EFFECTS OF SULFANILAMIDE: II. METHEMOGLOBIN FORMATION AND ITS CONTROL. A. F. HARTMANN, A. M. PERLEY and H. BARNETT, J. Clin. Investigation **17**:699 (Nov.) 1938.

A study was made of factors leading to the accumulation and disappearance of methemoglobin in patients to whom sulfanilamide was administered. Reasonably close agreement was found between the direct spectroscopic determination of methemoglobin and the determination of nonoxygen-carrying hemoglobin. In the majority of patients receiving 0.1 Gm. or more of the drug per kilogram per day cyanosis developed, and in every case of cyanosis the presence of methemoglobin was demonstrable. Methylthionine chloride (methylene blue) caused rapid disappearance of cyanosis with simultaneous reduction in the methemoglobin concentration in the blood when given intravenously in single doses of 1 to 2 mg. per kilogram or when given orally in doses of 65 to 130 mg., repeated every four hours. The latter medication also prevented any appreciable formation of methemoglobin when started simultaneously with the administration of sulfanilamide.

GUEST, Cincinnati.

EFFECTS OF HIGH HUMIDITY ON SKIN TEMPERATURE AT COOL AND WARM CONDITIONS. H. FREEMAN and B. A. LENGYEL, J. Nutrition **17**:43 (Jan.) 1939.

Data were obtained from a study of 10 normal adults at environmental temperatures of 24 and 32 C. (75.2 and 89.6 F.) and relative humidities of 20 to 90 per cent. When the environmental humidity is increased, the temperature of the skin is elevated, with higher recordings from the extremities than from the trunk.

FREDEEN, Kansas City, Mo.

DIFFICULTIES IN ABDOMINAL DIAGNOSIS IN CHILDREN—ILLUSTRATIVE CASES. D. JENNINGS, J. South Carolina M. A. **34**:282 (Nov.) 1938.

This is an excellent paper which is well worth any physician's or pediatrician's time. The various abdominal conditions which might occur and be confused as a picture of appendicitis are discussed, and several cases are outlined. The condition which are emphasized are Meckel's diverticulum, intussusception, renal and ureteral colic, pyelitis, arachnidism, acute mesenteric lymphadenitis, sickle-cell anemia, pneumococcic peritonitis, intestinal parasites and acidosis. The confusion of the last condition with appendicitis is illustrated by a case. Jennings believes that many patients operated on for appendicitis really have acidosis. Stress is laid on thorough physical and laboratory examination, with sufficient delay to determine the exact diagnosis.

WESTON, Columbia, S. C.

SULPHANILAMIDE. B. DURIE, M. J. Australia **2**:1103 (Dec. 31) 1938.

Durie presents an excellent review of the historic and pharmacologic aspects of sulfanilamide.

GONCE, Madison, Wis.

USE OF SULPHANILAMIDE IN SOME CLINICAL CONDITIONS. J. CHESTERMAN, M. J. Australia **2**:1110 (Dec. 31) 1938.

Chesterman discusses the clinical use of sulfanilamide.

GONCE, Madison, Wis.

INHALATION THERAPY AND ULTRAVIOLET RAYS IN PREVENTIVE PEDIATRICS. A. M. NARDI, Méd. inf. **10**:15 (Jan.) 1939.

Inhalation therapy, consisting of the introduction into the body through the respiratory tract of a medicament as a gas or as a mist, was reported by Hippocrates as early as 470 B. C. Celsius, Pliny and Galen also reported this method of treatment, the last recommending for certain diseases the breathing of air containing sulfur from the neighborhood of Mount Vesuvius.

Herodotus, about 500 B. C., recorded the therapeutic value of solar rays. Cornelius, Celsius and Galen later reported the beneficial action of the sun on the health.

A short review is given of the present day use of these two kinds of physical therapy.

HIGGINS, Boston.

NONINFECTIOUS ELEVATION IN TEMPERATURE AMONG ADOLESCENTS. W. I. MOLTCHANOW, Rev. franç. de pédiat. **13**:155, 1937.

Moltchanow concludes that slight elevations of temperature over a long period may be evidences of a neuropathic makeup or an allergic phenomenon. Higher temperatures are often simulated, the child wanting attention or freedom from school. Careful observation of the child while the temperature is being taken is necessary. The treatment consists in helping the patient make a better adjustment to the environment.

LESLIE, Evanston, Ill.

FAVISM IN CHILDHOOD. E. INGLESSI, Riv. di clin. pediat. **36**:1075 (Dec.) 1938.

A child 15 months old showed hemoglobinuria and marked anemia about twenty-four hours after ingesting a small quantity of cooked dried beans. In spite of subcutaneous injections of blood and the giving of stimulants, death ensued three days later.

HIGGINS, Boston.

AVENUES OF ABSORPTION OF MAGNESIUM SULFATE AND ITS CLINICAL APPLICATION. J. R. FERNÁNDEZ CANEPA, Rev. méd. latino-am. **23**:1414 (Sept.) 1938.

Fernández Canepa reviews the various methods of administering magnesium sulfate and the therapeutic results. In 20 to 25 per cent concentration it is useful in pertussis, the dose being 2 to 4 cc. Its sedative action makes it valuable in puerperal eclampsia, neuropathies, extrasystoles, tachycardia, asthma, chorea and tetanus. In premedication anesthesia a 50 per cent solution is given in 2 to 3 cc. doses intramuscularly. It is a depressant of the circulatory and respiratory centers, especially of the latter. It is contraindicated in cystitis, nephritis and meningitis.

DWYER, Kansas City, Mo.

IDENTITY OF THE TWO HANDS IN ONE SISTER AND EXTREME DISSIMILARITY OF THE TWO MEMBERS IN ANOTHER. SIEGERT, Fortschr. a. d. Geb. d. Röntgenstrahlen **56**:439 (Sept.) 1937.

In one sister the morphologic identity of the bones of the two hands as revealed by the roentgenogram was impressive; even the normal degree of asymmetry was lacking. Another sister, 2 years older, showed a congenital structural anomaly of the left hand which resulted in marked disparity between the two members.

MCCUNE, New York.

Society Transactions

UNIVERSITY OF MICHIGAN PEDIATRIC AND INFECTIOUS DISEASE SOCIETY

DAMON WALTHALL, M.D., *President*

Seventeenth Annual Meeting, Ann Arbor, Mich., Nov. 11 and 12, 1938

D. MURRAY COWIE, M.D., *Secretary-Treasurer*

POSTLACTATION FEEDING: PRESIDENT'S ADDRESS. DR. DAMON WALTHALL, Kansas City, Mo.

Breast milk as the sole food is adequate for only a short period, four to six months; before this time it must be supplemented by other foods if a perfect nutritional result is to be assured.

To maintain lactation, regular and thorough emptying of the breast is necessary. Nursing is the best stimulation; next is the electric breast pump, and manual expression is probably equally efficient.

By heredity some mothers are milk producers and others fat and beef producers. The latter trait is upsetting to the young modern mother; she can produce no milk; the mental and physical hazards of getting fat block all her chances. If her state of mind has ruled out the desire to nurse her baby, the problem is discouraging. Such mothers never supply sufficient breast milk.

Two procedures in the obstetric service favor drying the breasts: the daily weighing of the baby and too early starting of supplementary feeding with a so-called stock formula. The baby comes to the breast with no hunger but with a gain in weight; the result is secretory failure. The pediatrician now enters and is given credit for taking the baby off the breast.

A sore, painful and sometimes abscessed condition of the breast, which frequently follows a sore, painful, fissured state of the nipple, is the greatest individual factor in preventing a good flow. Menstruation or an interloping pregnancy should not be an indication for weaning.

Contraindications to maternal nursing are complete absence of milk by the end of the second or third week following early gushing and engorgement of the breast, an intractable inverted painful state of the nipples, previous operation for mastitis, excessive fat or benign tumors and a contagious or infectious disease in full swing at the time of delivery.

A second group of conditions mean relative contraindication. When the baby shows no evidence of syphilis and the mother has a recent active syphilitic infection, the baby should not be nursed. With typical congenital syphilis, if it is known that the mother also has the disease, the baby may be nursed; when the mother has anemia, is underweight, is nephritic or has heart disease, pneumonia or epilepsy, the baby should not be nursed. If the mother has a common cold, streptococcic sore throat or influenza, in particular, great precautions should be taken to protect the infant, even to the point of weaning. If the baby has long crying periods at night and does not sleep during the day, has enterospasm, an allergic phenomenon or an absolute intolerance to the mother's milk, weaning is necessary. With such conditions babies do well on *Eiweiss,* or protein, milk. The breast milk station comes to the aid of certain premature, weak or sick babies.

The basis of all bottle feeding is preferably boiled fresh cow's milk, with the addition of water and a consistent amount of carbohydrate, except when a special formula or evaporated cow's milk is necessary. These preparations, in my experience, do not lend themselves well to being modified or adapted to the baby's individual needs.

In a liquid milk formula the fat content may be varied from none to as much as the individual needs demand, and the curd may be varied to almost any suitable tension. Sugar is the only more or less constant food element. Usually if a baby is kept on a good formula long enough he will adapt himself to it.

Physicians cannot accept with complacence the fact that more and more artificial substitutes for maternal nursing are being used. The frequent necessity for giving infants other food than breast milk is not usually accompanied by failure. Success, in fact, is the rule. Lamb stated: "It is quite proper to encourage breast feeding, but we should not make the mistake of disfavoring substitute feedings, for . . . the babies fed on artificial milk formulae have shown no serious deviation from the normal development that regularly follows adequate breast feeding." This states my own feeling in the matter.

AN ATMOSPHERE OF REDUCED NITROGEN WITH RESPIRATORY OBSTRUCTIONS. DR. CLEMENT A. SMITH, Boston.

It has been possible in several different ways to alter the atmosphere which a patient breathes or the pressure under which it is taken into the lungs, so as to overcome asphyxia and anoxemia from respiratory causes. If asphyxia is due to weakness of respiratory muscles with a normal airway and normal pulmonary epithelium, relief may be obtained by the Drinker respirator. In the presence of a normal airway and normal respiratory musculature but a deficiency in permeability of the pulmonary epithelium, as with pneumonia, relief is obtained by altering the percentage of oxygen in the atmosphere breathed. This slightly increases the weight of the atmosphere. Thus if ordinary air, which is one fifth oxygen and four fifths nitrogen, is assigned an absolute weight of 1, air in which the oxygen tension is increased to one-half the total tension will have a relative weight of 1.05. Barach suggested the use of helium and oxygen mixtures in order to overcome the factor of weight. Such mixtures should pass through a narrowed airway and may even diffuse more easily across the pulmonary epithelium than oxygen and nitrogen mixtures because of the relative lightness of helium. A mixture which is 30 per cent oxygen and 70 per cent helium will weigh relatively 0.43. Such mixtures have been demonstrated by Barach to bring relief when the respiratory obstruction is in the main airways.

Paul Bert, in 1886, conducted studies with animals at low atmospheric pressures which showed that no matter how low a total atmospheric pressure was produced (specially constructed chamber), so long as the amount of oxygen present produced a pressure equal to the partial pressure of that gas in ordinary air, animals would breathe the mixture without difficulty. Wilson, Butler, Smith and others have reported studies made with a chamber in which such a condition is artificially produced: there is a diminished total pressure of atmosphere but a normal partial pressure of oxygen. Such a mixture, if the total pressure is reduced to 250 mm. of mercury and the oxygen pressure is maintained at the 150 mm. level of normal air, will weigh relatively 0.35 as against the weight of 1 for a unit of normal atmosphere containing the same amount of oxygen. The difference is of course due to a marked reduction in nitrogen. Such an atmosphere should pass an obstructed airway certainly as easily as a helium-oxygen mixture and perhaps should diffuse more easily through inflamed pulmonary epithelium.

In general the following observations were made. 1. In an atmosphere of low nitrogen and normal oxygen tension there is definite mechanical relief from artificial obstruction of respiration. 2. It is extremely difficult to prove this effect by studies of arterial blood, as it was found that for almost any degree of obstruction the resulting compensation by increased respiratory effort and increased volume flow of blood kept the arterial blood at or near normal levels of oxygen saturation. To study the relief of obstruction, therefore, it was necessary to work with samples of alveolar air from human subjects. The data obtained indicated that low nitrogen mixtures were at least as satisfactory as helium-oxygen mixtures as a means of relieving such obstruction. Actual saturation of arterial blood

under conditions of low nitrogen was somewhat better than might have been predicted, a finding which indicates possibly greater diffusion of oxygen in this form through the pulmonary epithelium.

Because of the highly experimental state of the project, no work has yet been done on patients, and no findings on disease conditions are reported.

DISCUSSION

DR. HARRY A. TOWSLEY, Ann Arbor, Mich.: Did you make any observations on the carbon dioxide–combining power of the blood and on the alkali content of the urine? What was the relation to the carbon dioxide content of the alveolar air?

DR. CLEMENT A. SMITH, Boston: A few determinations showed no particular differences with the atmosphere of reduced nitrogen from what would be expected with room air.

DR. DAMON WALTHALL, Kansas City, Mo.: Do you think this method of treatment can be developed into a practical method for the care of patients with respiratory obstruction?

DR. CLEMENT A. SMITH, Boston: Your question is difficult to answer. This therapy is still in a decidedly experimental stage. I should say the expense involved would make it unlikely that a chamber such as is described would be of wide usage. It is to be hoped that by its use discoveries may be made which will help in caring for patients with respiratory obstruction.

SEQUELAE OF POSTINFECTIONAL ENCEPHALITIS, WITH SPECIAL REFERENCE TO MENTAL INVOLVEMENT. DR. LOUISE SCHNUTE and MRS. E. W. MALLORY, Ann Arbor, Mich.

It has been known for many years that serious nervous manifestations may follow or accompany any of the infectious diseases of early childhood. It appears that residual effects of measles, pertussis and smallpox vaccination have been more frequently associated with encephalitic changes than were noted formerly. A number of these patients have been seen in our clinic. Impressed by the encephalitic sequelae, we have felt that they should be more thoroughly studied, to discover a possible relationship between the infectious diseases, emotional instability and mental retardation.

At this time we are attempting not to draw conclusions but only to present a preliminary report. Disease histories have been compiled and psychologic data collected for 85 children, 13 years old or younger, who were seen between July 1 and Oct. 1, 1938. Forty-nine of these were outpatients and 36 house patients. The psychologic tests employed were Terman's Old Form Scale, the L Form Stanford-Binet and, for very young children, the Kuhlmann. Thirty of the 85 children were referred by departments other than the pediatric, i. e., by the departments of neurology, social service, orthopedics, dermatology and psychotherapy. For about 21 of this group, no disease histories were available. For the remaining 64 patients, chosen from those whose records presented disease histories, we obtained the following data:

Disease	Percentage of Group
Measles	69
Mumps	31
Pertussis	56
Chickenpox	41
Poliomyelitis	1.6
Encephalitis	1.6
Convulsive disorders	19
Scarlet fever	14
Pneumonia	12.5
Diphtheria	1.6
Rickets	3
Paraplegia	8

Since these patients were not tested before their illness and there is little record of their intellectual advancement prior to their illness, it has been impossible to discover the time of the beginning or the rate of mental retardation. We are therefore handicapped in establishing an actual relationship between the mental retardation and the encephalitic processes. The parents frequently reported emotional instability or mental dulness to have been coincident with or to have followed closely an infectious disease.

It seems worth while to continue this study for the purpose of securing more specific information regarding a larger number of cases, meanwhile placing emphasis on recording more detailed histories and more definite data relative to the beginning of emotional and mental changes following the so-called diseases of childhood.

DISCUSSION

Dr. Harold Rothbart, Detroit: In only 1 case, I believe, was autopsy performed. This sequela occurs more frequently than physicans are able to detect clinically. I believe it is more frequent than brain tumor. There were no symptoms of brain tumor in this case before measles developed, but brain tumor sometimes occurs after measles. Possibly measles precipitated the development of a tumor.

Dr. Rockwell Kempton, Saginaw, Mich.: I congratulate Dr. Schnute on the innovation of presenting clinical cases. One would like to see more of this.

Mrs. E. W. Mallory, Ann Arbor, Mich.: My interest in attempting to discover a relationship between mental retardation and infectious diseases is that of a layman who has found among fathers and mothers an appalling disregard of the possible serious effects of measles, mumps and other so-called diseases of childhood. We hope that a study of the history and of the sequelae in such cases will produce results which will quicken parents' understanding of the danger of mental as well as physical affliction. For it is through such knowledge that they will be induced to seek medical aid promptly, isolate patients on suspicion and observe strict quarantine to protect their own and others' children from grave misfortune.

Hypothyroidism in Children. Dr. Franklin J. Mellencamp, Ann Arbor, Mich.

The case histories of 73 children, seen in the past three years, whose condition was diagnosed as hypothyroidism are reviewed.

The criteria for the diagnosis include (1) a basal metabolic rate below —10 per cent; (2) a blood cholesterol level above 250 mg. per hundred cubic centimeters, and (3) retarded ossification as evidenced by roentgen demonstration of epiphysial appearance and closure.

The limitations of these criteria must be regarded; for example, malnutrition, edema and pituitary and adrenal disorders are associated with low metabolic rates; determinations of cholesterol, too, often show no relation to the basal metabolic rate, and hypothyroidism of short duration exhibits no changes in the osseous system.

Obesity was the most prevalent presenting complaint (42 per cent). Eight per cent of the group were underweight. Constipation and vague abdominal pain were the chief gastrointestinal symptoms. Mental retardation was recorded for 26 per cent; those having initial and subsequent determinations of the intelligence quotient showed no appreciable respose to thyroid medication. Fifteen per cent were regarded as mentally unstable. Cutaneous manifestations of some sort were described for 9 per cent; their significance is questionable. A possible relation between certain orthopedic conditions and hypothyroidism may be drawn; flat feet, Perthes' disease, slipped femoral epiphysis and knock knees were noted in 14 per cent. Some allergic manifestation was found in 20 per cent; this falls within the accepted incidence in the general population. Endocrine relationships are difficult to evaluate; possibly thymic enlargement is related. Thyroid enlargement

was present in 8 per cent; hypogenitalism, cryptorchidism and menstrual disturbances were included. In our series Fröhlich's syndrome in 2 instances, and a suprasellar tumor were erroneously diagnosed as hypothyroidism.

There were 2 particularly interesting cases: A 10 year old boy with hypothyroid findings but presenting the unusual mental manifestation of delusions responded well to thyroid medication. The basal metabolic rate was —26 per cent on initial determination. A 12 year old girl with no hypothyroid manifestations complained chiefly of dysmenorrhea and malnutrition. On admission her weight was 76 pounds (34.5 Kg.) and her basal metabolic rate —29 per cent. Administration of thyroid alleviated the pain and apparently was instrumental in causing a gain of 19 pounds (8.6 Kg.) in two months.

The diagnosis of hypothyroidism in children, as in adults, apparently demands the cognizance of the unusual as well as the classic manifestations of the condition. No one criterion can be taken as the basis for the diagnosis unless the limitations of that finding are considered. Untoward effects of thyroid with nonhypothyroid conditions are uncommon. The use of this substance as an aid in the diagnosis of borderline cases seems advisable.

DISCUSSION

Dr. John L. Law, Ann Arbor, Mich.: If determinations of cholesterol were made for the younger age group, would they not be of diagnostic value?

Dr. Franklin J. Mellencamp, Ann Arbor, Mich.: As an index of thyroid function in the group of children under 7 years, determinations of blood cholesterol seem to be of value, but the results in this limited group were disappointing.

Dr. Clement A. Smith, Boston: Were basal metabolic rates of —1 per cent included in your series, or is the figure in your chart in error?

Dr. Franklin J. Mellencamp, Ann Arbor, Mich.: That was the first determination listed. The subsequent rating was —11 per cent.

Dr. King (by invitation): It seems to me that it is important to know the type of thyroid preparation used. There is marked variation in the potency of the available products. For instance, ¼ grain (0.016 Gm.) of one manufacturer's thyroid, in my experience, did more than some other preparations.

Dr. Harold Rothbart, Detroit: Roentgen findings are not entirely reliable in determining mental deficiency. In this group there may be roentgen evidence, but a physician does not "get to first base" by treating these patients with thyroid extract. The hypothyroid child is not necessarily mentally deficient.

Dr. Campbell Harvey, Pontiac, Mich.: How about the therapeutic test for patients with menorrhagic conditions?

Dr. Franklin J. Mellencamp, Ann Arbor, Mich.: This is often a worth while procedure, even in the absence of the usual criteria of hypothyroidism.

Dr. Harold Rothbart, Detroit: Thyroid medication stimulates the whole organism.

Dr. Franklin J. Mellencamp, Ann Arbor, Mich.: Apparently several clinical pictures may be characteristic of so-called masked hypothyroidism. Cognizance of this fact and of the several criteria of hypothyroidism will lead to satisfying results in a limited number of cases.

Gross Intestinal Hemorrhage Due to Pathologic Condition in Meckel's Diverticulum in Two Infants Under One Year of Age. Dr. Mark L. Osterlin, Traverse City, Mich.

Meckel's diverticulum is relatively uncommon. It occurs in from 2 to 4 per cent of the population. In only about 25 per cent of the cases are pathologic changes noted. Two patients under 1 year of age were seen in Central Michigan Children's Clinic during the past year.

R. G., aged 10 months, was referred because of bleeding from the rectum. Data on the child's family, birth and development were irrelevant. Breast feed-

ing was continued three months, then similac was given for six months. The appetite was good; the bowels were regular. Eczema had been present since the patient was 3 months old. At 7 months several ounces of bright red blood were passed by rectum. A second hemorrhage was noted one and a half months later; a third hemorrhage occurred one week before the child entered the clinic. The stools were otherwise normal.

Physical examination showed a well nourished infant; findings were essentially negative. The abdomen was entirely normal. There was mild eczema of the face and the flexure surfaces of the arms. Reactions to the Kahn and the tuberculin test were negative. Three days later the baby was admitted to the hospital because of restlessness and irritability. The examination otherwise gave negative results. The bleeding time was five minutes and the clotting time four and one-half minutes. The hemoglobin content (Sahli) was 65 per cent. There were 3,900,000 red cells and 20,600 white cells with 40 per cent polymorphonuclears, 57 per cent lymphocytes and 3 per cent eosinophils. The urine was normal. Examination of the stool showed occult blood. Five days after the patient's admission a laparotomy was done, and a red, inflamed Meckel's diverticulum with ulceration was removed. The postoperative course was uneventful. The diverticulum measured 3 by 1.5 by 1 cm. A hemorrhagic area could be seen in the mucosa. Microscopically, there was ulceration of the mucosa with marked polymorphonuclear infiltration of all layers. There were congestion and edema of the serosa and a few areas of hemorrhage in the submucosa. No heterotopic mucosa was found.

T. F. was seen at the age of 11½ months. Data on the patient's family, birth and development were irrelevant. At the age of 6 months a profuse hemorrhage from the rectum was noted. Examination of the blood on that date showed a hemoglobin content of 58 per cent, 2,800,000 red cells, 8,000 white cells and 300,000 platelets. The bleeding time was six and one-half minutes and the clotting time two and one-half minutes. Two hundred and twenty-five cubic centimeters of citrated blood was given intravenously. A barium sulfate enema showed no abnormality. A tentative diagnosis of hemorrhagic disease or Meckel's diverticulum was made. Three weeks later the hemoglobin content was 75 per cent. Six days later a black, bloody stool was passed. Results of a subsequent examination were negative. Twenty cubic centimeters of the mother's blood was given intramuscularly, and on the following day the hemoglobin content was 65 per cent. A capsule of a liver and stomach extract with iron (lextron) was given three times daily. One month later the hemoglobin was 85 per cent. About two months after this another gross hemorrhage from the bowel occurred, and blood was given intramuscularly. Thirty days later bloody stools were passed for about three days. The hemoglobin content at this time was 70 per cent.. In two weeks a recurrence of bleeding was stopped by intramuscular administration of blood. Six weeks later hemorrhage again was noted for two days; at this time the hemoglobin content was 52 per cent. The patient was referred to the Central Michigan Children's Clinic.

Examination showed that the infant was rather pale but otherwise appeared to be normal. Results of the physical examination, except for the anemia, were entirely negative. The abdomen was soft. No abnormal masses or areas of tenderness were found. Transfusion and surgical procedure were advised. These were performed on the following day, at which time a Meckel's diverticulum, 3 by 1 cm., was found 24 inches (61 cm.) proximal to the ileocecal valve. An ulcer 6 mm. in diameter and 3 mm. deep was located near the base. A clot of blood lay in the crater. The mucosa was grossly similar to that of the stomach. No pathologic studies were made. The postoperative course was uneventful.

DISTRIBUTION OF PNEUMOCOCCI IN EXPERIMENTALLY INFECTED RATS AND PENETRATION OF ANTISERUM INTO THE LESIONS. ALICE H. KEMPF and DR. W. J. NUNGESTER, Ann Arbor, Mich.

It has been generally recognized that antibacterial serums are not effective in the treatment of infectious diseases, particularly if treatment is not begun immedi-

ately. Antipneumococcus serum, either horse or rabbit, while effective if given early in the disease does not produce nearly such striking results in man or in the experimental animal if the administration of the serum is delayed.

In an attempt to understand this failure of late serum therapy, it was decided to study the distribution of pneumococci in the bodies of rats at various intervals following intrabronchial inoculation of pneumococci and mucin, with or without subsequent treatment. In untreated rats, forty-eight hours after inoculation organisms were present not only in the lungs but in the blood and in other organs such as the spleen, the liver and the kidneys. Treatment given at this time did not free these organs of bacteria and only temporarily cleared the blood stream. Treatment given twenty-four hours after inoculation, a time at which the lesion corresponded to a two or three day pneumonia in man, did not reduce the number of organisms in the lung but succeeded in keeping the blood and other organs relatively free of bacteria. Treatment at twenty-four hours did not decrease the mortality of the rats below 50 per cent; untreated controls had a mortality approaching 100 per cent.

Since the pneumococci were not decreased in the pneumonic lung, the question of penetration of the lung by antipneumococcus serum was next considered.

Although the limited occurrence of necrosis in pneumonic lungs seems to give evidence of patency of the blood vessels, it was decided to investigate this condition experimentally. A radiopaque substance, colloidal thorium dioxide, was injected intravenously, the lungs were removed, and roentgenograms were taken. It was found that the larger blood vessels were filled with the radiopaque material.

Rats were bled from the abdominal aorta and perfused with heparin. The left lobe was removed, and the pulmonary artery was cannulated. The volume flow of saline solution through the almost completely involved pneumonic lung was found to be 5 per cent of that of a normal lung. If a rat was given intravenous injections of methylene blue, the normal lung was dyed at once while only certain portions of the pneumonic lobe became blue. These experiments, with others, seemed to show that the pneumonic lobe was patent but that the rate of circulation was materially reduced in certain areas.

Demonstration of antipneumococcus serum, as such, by any method was found to be impossible in the pneumonic lung, owing to the large amount of pneumococcus polysaccharide present in the infected animal. It was estimated that the pneumonic lobe at twenty-four hours contained approximately 5 mg. of soluble specific substance, which was able to neutralize 25 to 30 cc. of antipneumococcus serum.

However, it was possible to examine the lung for the presence of antibody after the injection of rabbit hemolysin serum of high titer. Hemolysin was never demonstrated in the pneumonic lung, although the blood showed a titer in some cases of 1:2,560. Rarely, the normal lung showed the presence of hemolysin in a titer of 1:10.

From this work it seems that the action of antipneumococcus serum is in part that of keeping the blood and the organs other than the lung free of bacteria. There may also be an immediate effect on toxin-like substances. There is little reason to believe that the antiserum reaches the organisms in the alveoli of the infected lobe or that if it should penetrate it would have much effect because of the large amount of polysaccharide present. Further work is being done on the mechanism of the spread of pneumococci in the pneumonic lung to determine why serum given immediately after inoculation prevents the spread of the lesion in an experimental infection. The organisms in the older part of the pneumonic process are doubtless destroyed rapidly by the phagocytic cells, as shown by other workers. It seems likely, however, that serum is no more able to reach the advancing than the older portion of the lesion, and this presents a definite and grave disadvantage to the patient. A similar failure of antiserum to penetrate various tissues of the body is probably universal in the treatment of infectious diseases resulting from proliferation of bacteria at a local site.

DISCUSSION

DR. HAROLD ROTHBART, Detroit: How much serum is available and how much is neutralized by other body tissues?

DR. W. J. NUNGESTER, Ann Arbor, Mich.: Although we do not know the exact mechanisms by which antipneumococcus serum acts, the current practice of giving so much serum that an excess can be demonstrated in the blood of the patient seems most reasonable.

CORD BLOOD SERUM FOR PROPHYLAXIS AND MODIFICATION OF MEASLES. DR. HARRY A. TOWSLEY, Ann Arbor, Mich.

The value of placental extract for the prevention and modification of measles is well recognized. Not infrequently after the injection of this substance the child will exhibit certain general systemic and focal reactions. It is thought that these reactions are brought on by the presence of tissue proteins, which are contained in immune globulin preparations.

Goodall and others have demonstrated a technic for obtaining blood from the fetal cord at the time of delivery. It seems logical that the plasma of this blood should contain the immune bodies found in placental extract. Specimens of serum were obtained from cord blood by centrifugation. Several of these were pooled, tested for sterility and given as a prophylaxis for measles.

Fifty-two children (6 months to 12 years old) with definite known exposures to measles in the spring of 1938 were given intramuscularly from 8 to 10 cc. of cord blood serum within one to five days after exposure. In 5 of these signs and symptoms of measles developed; in each the disease was mild and of short duration. One received serum one day after exposure; 3, three days after exposure, and 1, five days after exposure. Two of those who contracted measles were in constant contact with siblings who had the disease.

No conclusions can be reached from such a small series of cases, and full cognizance has been given to the facts presented by Karelitz as to the evaluation of contacts.

DISCUSSION

DR. CLEMENT A. SMITH, Boston: How do you obtain the serum without getting tissue proteins? You have had a good yield from these placentas. I believe Dr. McKhann has obtained some reactions to placental extract which seemed to be due to tissue juices, but you apparently have observed no reactions.

DR. HARRY A. TOWSLEY, Ann Arbor, Mich.: There is little opportunity for tissue proteins to enter into this serum because no tissue is macerated, as it is in the preparation of placental extract. At the time of delivery, from 70 to 130 cc. of blood may be obtained from the placental cord. This yields from 40 to 50 per cent serum, or from 30 to 65 cc.

Open Forum

THE THYMUS AND THE THYMICOLYMPHATIC CONSTITUTION

INTRODUCTORY REMARKS. DR. D. MURRAY COWIE, Ann Arbor, Mich.

"The Myth of the Thymus" is the title of a recent paper from Ireland. But the last word on the problem of the thymus has not been said. All pediatricians are acquainted with a clinical syndrome believed to be of thymic or thymicolymphatic origin. It is known that the thymus is large at birth and becomes smaller as time goes on; it involutes. This is the normal process in childhood. It is known that the thymus becomes atrophied during the course of a wasting disease. This is the picture most frequently seen at autopsy. It is known that

the thymus is often seen to be enlarged and that the lymphatic structures are hyperplastic in many instances of sudden death, particularly of infants and children. Hammar stated (1926-1929) that in the large percentage of children who die accidentally the lymphatics are as large as they are in status lymphaticus. He stated the belief that pathologists are in error because, he explained, they have secured their information on the thymus and the lymphatic state from routine autopsy material, which represents the state after a prolonged illness. This has greatly influenced pediatric thought.

Disturbances of the thymus and the lymphatic structures must often be momentous. In certain animals excision causes general retardation of growth, affects calcium metabolism, causing calcemia, and causes genital hypoplasia and slight hyperthyroidism. Hypertrophy of the thymus is said to affect the endocrine function of the pancreas, and certain endocrine conditions of the body are thought to influence the process of thymic hypertrophy. Atrophy of the thymus in normal and in hypophysectomized rats follows the administration of extract of adrenal cortex.

There is evidence of a thymus hormone. If thymus extract is given to thymectomized rats, the retardation of growth caused by excision is overcome. Estrogenic substance, progestin and gonadotropic factor substance when administered to certain animals modify the histologic structure and the functions of the thymus. Antithymic and antigonadal precipitins have been found in normal guinea pig serum. There seems to be a direct relation between these precipitins and the development of the thymus and the gonads.

Stridulous laryngitis is thought by some to be of thyrothymic origin. It is thought that under certain conditions so important a condition as the acid-base balance is influenced by the thymus. It is said that changes in the p_H of the urine may be induced by thymic disturbance. Physicians are much aware that convulsive movement, stridor and cyanosis may be due to disturbances not usually attributable to the thymus—for example, tetany, which responds to calcium therapy. It is known that calcium deficiency may be due to disturbances other than those of the thymus.

It is known that roentgen irradiation decreases the size of an enlarged thymus and stops the so-called thymic attack, and it is believed that it stops the occurrence of subsequent attacks. As a prophylactic, it is supposed to prevent the development of an attack occasioned, for example, by an anesthetic. Many, if not most, physicians in the East think that those in the goiter belt are "all wrong on the thymus." Those who leave the goiter belt and go east to live often change their minds about the goiter problem. They either no longer see the familiar syndrome or attribute it to some other cause. Years ago experience led physicians to fear the giving of an anesthetic to a child thought to be of the thymicolymphatic type. Many deaths in young children were thought due to the thymicolymphatic state. Prophylactic preoperative roentgen irradiation was introduced. Fatalities have apparently been fewer since this method was adopted. The possibility that allergy plays a part has been suggested.

In the light of advanced knowledge concerning the thymus, it is desirable to reopen this so-called closed question.

Shall physicians continue to irradiate the thymus preoperatively in young children?

CLINICAL ASPECTS AS SEEN BY THE OBSTETRICIAN. DR. NORMAN F. MILLER, Ann Arbor, Mich.

While the obstetrician is greatly interested in all aspects of so-called thymic disease, his immediate concern is confined to the neonatal period. Certainly there is no unanimity of opinion regarding the significance of an "enlarged thymus" during this period and during the first few months of the infant's life. In large measure the attitude of any hospital group is determined primarily by the

pathologist and secondarily by the pediatrician and the roentgenologist. The pathologist's attitude at the autopsy table has considerable bearing on the group attitude toward thymic enlargement and its significance. It is high time for some effort to establish more definitely the role—if any—of an enlarged thymus in the health of a newborn child. To this end the following questions seem pertinent:

1. What is the accepted normal size of the thymus in a newborn infant? Too much variation exists in this respect. What is considered normal by some (15 to 20 Gm.) is looked on as abnormal by others. Certainly the gland is larger in the well nourished than in the poorly nourished child. Can there be, therefore, a hard and fast statement as to normality without considering the infant's weight as well?

2. Shall we assume that so-called thymic death is based on status thymicolymphaticus or is it a matter of one organ? If it is based on a lymphatic constitution, is irradiation of the thymus gland adequate or desirable?

3. In view of the many factors which influence the size of the thymic shadow, can any real diagnostic value be placed on the average thymic roentgenogram?

4. If radiation therapy is of value, how soon after treatment may such therapy be considered effective? Does it do any harm?

It is of course an easy matter to ask questions, but since there appears to be little doubt in the minds of some regarding thymic enlargement as an entity, perhaps the answers will be relatively easy.

CLINICAL ASPECTS AS SEEN BY THE OTOLARYNGOLOGIST. DR. ALBERT C. FURSTENBERG, Ann Arbor, Mich.

It would be presumptuous to say that an enlarged thymus is responsible for the thymicolymphatic constitution, although I believe one occasionally observes a definite symptom complex which is associated with enlargement of the thymus and which causes death of children. In many instances, the pathologist has demonstrated an enlarged gland when there have been clinical evidences of a thymicolymphatic constitution prior to death.

The term "thymicolymphatic constitution" is out of favor here and abroad and is said by some authorities to be a "figment of the imagination of the otologist and the pathologist." There exists, however, a condition characterized by definite clinical features which causes death, and the pathologic features constitute an entity which is termed "thymicolymphatic constitution." The syndrome presents the following clinical aspects:

1. *Stridor.*—There is evidence of embarrassed breathing characterized by dilatation of the alae of the nose, prominence of the sternocleidomastoid muscles and retraction of the epigastrium and of the intercostal spaces. Frequently this stridor is termed "congenital laryngeal stridor," which is an entirely different entity due to organic changes clearly visible by direct laryngoscopic examination. Congenital laryngeal stridor is due to certain changes in the epiglottis and the larynx: absence of cartilage in the former and collapse of the soft tissue due to inadequate support in the latter. Thus the epiglottis falls over the lumen of the larynx, producing respiratory embarrassment. Infants afflicted with this condition usually survive until the cartilages grow or take on sufficient resilience to lift the epiglottis and restore the lumen.

2. *Syncope.*—The picture is that of collapse, with ashen pallor of the skin and rapid, feeble pulse but seldom any indication of cyanosis. I have grave doubts that the cause of death is mechanical pressure on the trachea. The signs are not those of tracheal obstruction.

The following theories as to the cause of death may be mentioned:

1. *Compression of the Trachea.*—If serial anatomic sections of the neck and thoracic cage are studied, one discovers that the thymus gland does not overlap

the trachea to any great extent. It is situated low in the mediastinum, overlaps the pericardium but is in apposition to the trachea for only a short distance above the heart. It requires a weight of approximately 110 Gm. to compress the trachea of a child 5 years old. The average weight of the thymus is 24 or 25 Gm.; some have weighed 40 to 45 Gm., and one, reported by Dr. Weller, weighed 126 Gm. I do not believe that one frequently encounters a thymic structure of sufficient size and weight to cause complete obstruction of the trachea.

2. *Pressure on the Vessels of the Neck.*—This would cause congestion and cyanosis of the head and neck, but these signs are not usually seen.

3. *Pressure on the Nerves in the Neck.*—This would cause paralysis of the recurrent laryngeal nerve. I have examined larynxes of children suffering from thymicolymphatic constitution and have found them perfectly normal, without evidence of laryngeal paralysis.

4. *Anaphylactic Change.*—My theory as to the cause of death is based on the following observation: A child 6 years old was brought to the clinic with a history of foreign body aspiration. The description of a sudden choking seizure followed by a period of comfort was so typical that I inserted the bronchoscope in search of the foreign body. The larynx and trachea appeared normal, and the bronchoscope was passed through these structures directly into the right primary bronchus. The patient suddenly died with the bronchoscope in this position. The mucous membranes of the bronchi became swollen and edematous and gave the appearance of a sudden severe allergic reaction. The picture was so vivid and dramatic that I was perfectly certain that the cause of thymicolymphatic death had at last been revealed to me. An autopsy was performed, but much to my surprise the mucous membranes of the bronchi were found perfectly normal. I can only conclude that in thymicolymphatic constitution some kind of an anaphylactic change occurs which obstructs the bronchi and disappears after death.

ROENTGEN FINDINGS. DR. VINCENT C. JOHNSON, Ann Arbor, Mich.

By roentgen examination it is possible to estimate the relative size but not the exact dimensions of the thymus in infants and children. The findings may be reported as "no enlargement," "moderate enlargement," or "marked enlargement."

Normally the thymus of a young person is largely hidden by the spine in the frontal projection, while laterally it is indistinguishable from the shadow of the great vessels. Variations in size related to respiration are readily recognized by the aid of the fluoroscope. During expiration the upper part of the mediastinum appears broadened, and thymic enlargement may be erroneously reported.

Reliable examination for determination of thymic size should include four exposures: two during full inspiration and two during full expiration, the frontal and the lateral projection being represented in each phase. Enlargement to one or both sides of the shadow cast by the great vessels, particularly during inspiration, with deviation or narrowing of the trachea strongly suggests abnormal enlargement. In the lateral projections sharp angulation of the trachea and partial or complete obliteration of the normally clear anterior mediastinal space permit the same inference.

The exposures described must be carried out with technical perfection. Rotation, faulty timing with respect to the respiratory phase or lengthy exposures permitting undue motion promptly defeat the purpose of the examination.

If thymic death can occur as the result of mechanical pressure, the roentgenologist can render twofold service by recognizing abnormal thymic enlargement and by producing shrinkage of the thymus with small doses of external radiation: The required dose is far below the amount necessary to produce erythema in the skin.

Since it is definitely possible to describe the relative size of the thymus on the basis of roentgen findings, the roentgenologist should continue to report his obser-

vations, irrespective of clinical aspects, expecting that they will be properly evaluated by the clinician; he may confidently promise relief of pressure as the result of irradiation therapy.

DISCUSSION

DR. GEORGE KAMPERMAN, Detroit: Pediatricians vary in their opinion regarding this question, and obstetricians usually leave to them decisions concerning the thymicolymphatic constitution. One formerly spoke of "enlarged thymus" and "status lymphaticus"; now the terms are combined, though the conditions do not always go together. Is the adenopathy produced by the thymus or by some other factor? There are instances of symptoms due to compression of the air passages by an enlarged thymus. In these cases the baby has spells of cyanosis and stridor which improve on irradiation therapy. Has the baby laryngeal stridor or symptoms from the mechanical obstruction without the typical constitution? Roentgen diagnosis of enlarged thymus is common. If the patients with enlarged thymus all evidenced status thymicolymphaticus, and if they were all likely to die suddenly from trivial causes, would not more deaths from trivial causes be seen? Some roentgenologists have stated that there is no correlation between the size of the roentgen shadow, the postmortem observations and the symptoms. One may wonder if any enlargement is important unless it is rather extreme.

Some years ago I delivered a baby with an enlarged thymus. Radiation reduced the size of the thymus, but there was no decrease in the symptoms. At the age of 6 months the child came under the observation of Dr. Canfield, who found that a congenitally small larynx was responsible for the symptoms.

The roentgen report of enlarged thymus usually ends the investigation, and the baby is promptly given roentgen treatment. Other possible diagnoses must be considered.

In cases of so-called status thymicolymphaticus, why does it do any good to give the thymus roentgen therapy if the trouble is probably in the adrenals or if the enlarged thymus is only a small part of the picture? Why should one obtain good results from treating a small portion of the pathologic area?

According to Edith Boyd, the thymus shrinks quickly with inanition, febrile diseases and long illness. It is the thymus shrunken by previous disease that the pathologist usually sees. Dr. Boyd made the suggestion that the organ should be studied in patients who had had sudden accidental deaths. She found a fairly large percentage of instances of enlarged thymus associated with other lymphadenopathies or with evidences of these conditions. She expressed the suspicion that pathologists do not recognize the normal thymus in a person who has died without previous illness and that the so-called status lymphaticus is a normal condition in a person who has not been ill.

A pregnant woman presents no evidences that will foretell whether her baby will have an enlarged thymus. Miller, in 1924, pointed out that older women seem to have babies with this constitution more often than younger women, but my experience has not borne this out.

It has been suggested that there is a familial tendency to enlarged thymus; this seems doubtful.

I believe that there are cases in which the thymus is enlarged, but they are rather rare, and I am skeptical as to whether the roentgen evidence of an enlarged thymus is the basis on which one could prognosticate the possibility of a subsequent sudden death.

DR. EARL W. MAY, Detroit: In 1923, shortly after I took charge of the newborn service in the maternity division of the Herman Kiefer Hospital, a 7 day old American boy, weighing 11½ pounds (5,216 Gm.), was found dead in his crib. Autopsy showed a thymus larger than the heart, which impinged on the heart, the large vessels, the trachea, the large bronchi and probably the recurrent laryngeal nerves. Because of its size and thickness its upper poles extended to the thyroid gland.

In the course of two weeks another baby (5 days old) began to have projectile vomiting. It seemed too early for pylorospasm or pyloric stenosis. Gastrointestinal studies gave negative results. The roentgenograms, which included the chest, showed a large platter-like shadow in the upper part of the mediastinum. Roentgen treatment was given, and the symptoms subsided. Two more treatments were given at weekly intervals, and roentgen examination one week later showed complete disappearance of the thymic shadow.

Three weeks later a well developed 12 pound (5,443 Gm.) Negro girl, the sixth child, was found dead in her crib. The other 5 children were boys; they were living and well, but all had weighed less than their sister at birth. Autopsy revealed a large, thick thymus weighing 35 Gm., pressing directly on the trachea, large vessels and nerves.

After these three episodes it was ordered that all infants born in the maternity division should be examined roentgenographically in the anteroposterior position for study of the thymus. One hundred and fifty newborn infants of the indigent class, premature, weakling or normal and born at full term were examined. A distinct shadow in the upper part of the mediastinum beyond the lateral processes of the spine was demonstrated in the infants who were clinically considered to have thymic enlargement. The degree of enlargement was designated as 1, 2, 3 or 4 plus. The greatest enlargement was found in the following infants: (1) those whose birth weight was 8 pounds (3,629 Gm.) or over, (2) those of multiple births, (3) premature infants and (4) those with congenital anomalies such as harelip, cleft palate, cataract, extra digits, hemanigomas and spina bifida.

A standing order was then made that all babies in these classes except the premature, the severely ill or those suspected of cerebral hemorrhage should be examined roentgenographically within twenty-four hours after birth. The films were examined at the maternity pavilion, and the number of treatments to be given was designated after personal observation of each baby. This gave an opportunity to observe symptoms which some infants portray during the first twelve days of life, for example, cyanosis, breath holding, brassy cry, irritability and fright when being bathed, congestion or cyanosis of the face and mottling of the skin.

Some babies with a 4 plus shadow needed five or six treatments for proper reduction. Premature infants were examined as soon as they were able to be taken to the roentgen room.

This method was continued for two years without a thymic death, when a baby weighing 7 pounds (3,175 Gm.), with a brassy cry and intervals of cyanosis, showed no thymic enlargement but died the day after examination. A thick, narrow thymus, of the shoehorn or wedge-shaped type and weighing 27 Gm., was found at autopsy; apparently this death was caused by compression.

Since using combined anteroposterior and lateral technic, we have not lost a baby from thymic enlargement in the newborn service, and nearly 25,000 have been born there within the past fourteen years. I have seen many babies who had a thymic death without roentgen treatment; I believe they could have been saved if treatment had been instituted a few days before death. No newborn baby or infant up to 6 months of age who has the symptoms described should be prevented from having a roentgen examination and the treatments deemed necessary. This is good insurance and good prophylactic medicine.

I believe that there is a relationship between the thymus and the thyroid, because since the introduction of iodized salt in the goiter belt lessening of the number of cases of enlarged thymus has been coincident with lessening of the number of cases of goiter. A pregnant woman using iodized salt will have a child with a thymus of more normal size.

This report represents part of a clinical research program I have been slowly working on for the past sixteen years. I believe that a definite, large scale clinical investigation should be made regarding the thymus and the thymicolymphatic constitution. It should be carried on in the goiter belt, particularly in the Great Lakes area, and not where this condition is not commonly seen.

DR. SAMUEL W. DONALDSON, Ann Arbor, Mich.: Three age periods should be considered: the newborn period, the tonsil and adenoid period and the adolescent period. In every examination it is valuable to study the entire respiratory tract, including the posterior part of the pharynx, the trachea and the tongue in anteroposterior and lateral views. In older children, before either negative or positive findings are reported, one should rule out the presence of a foreign body, posterior pharyngeal abscess, pneumonia and other conditions that might be responsible for the symptoms.

The broad thymus shadow may be thin, flat, deep or narrow. If there is a thymic hormone or if the thymus in any way causes a condition similar to anaphylactic shock, the large flat thymus may be able to cause as much trouble as the narrow thymus which shows evidence of compression. Severity of symptoms does not correspond exactly with the size of the shadow. The symptoms cannot always be correlated with the roentgen findings.

I do not believe that treatment of status lymphaticus by irradiation of the thymus only delays the likelihood of a fatal episode. The large thymic shadow is probably the only demonstrable manifestation of such a constitution at the time the child is examined. Treatment evidently does affect the thymus at the time, but if the patient has that type of constitution, the disease will continue to develop as true status lymphaticus, and it is doubtful whether therapy over the thymus is of permanent value in arresting this further development.

Since the introduction of iodized salt, the incidence of enlarged thymus in this locality has decreased. Large thymus glands are not seen at autopsy as often as several years ago, probably because they are discovered early and treated. Towsley and I demonstrated that in approximately 1,500 newborn infants of mothers who had been using iodized salt for five years the incidence was much lower than that reported before the advent of iodized salt.

The constitution favorable to status lymphaticus is the heavy, overweight type. Early hyperplasia of lymphoid tissue is characteristic. Children with these features should have their tonsils and adenoids removed. Sudden deaths might occur, and, as would be expected, an enlarged thymus would be found. The circumstantial evidence appears to furnish a logical reason for surgeons to suspect that overweight babies and children are more likely to have an enlarged thymus. Some have advocated that all overweight babies be examined for an enlarged thymus.

In examining 2,000 consecutive newborn infants twenty-four hours after birth our standard technic included taking anterior-posterior and lateral views, making the exposure in inspiratory and expiratory phases and measuring at the level of the third dorsal vertebra, a width of 3 cm. being considered normal. Three and a half centimeters was considered borderline, and a greater width, with displacement and tracheal compression, positive indication of an enlarged thymus. Approximately half of the subjects were boys. An abnormally large shadow was seen in 11.3 per cent and some degree of enlargement in 18.4 per cent. Babies in the 8½ to 9 pound (3,643 to 4,082 Gm.) group showed the largest percentage of enlargement; 28 per cent showed some enlargement. The heaviest and the smallest of both sexes had no enlargement. In the heavier baby there is more increase of fat in the gland than actual increase of thymic tissue. The size of the thymic shadow is proportional to the size of the infant in those above average weight.

Since 100 roentgen units can be given to the thymus without any ill effects, I believe that in case of doubt it is better to administer a small amount of radiation than not to give any. A decrease in the size of the shadow is the rule. No deaths have occurred which could be attributed to the thymus in the group we have recently studied.

DR. GEORGE WALDBOTT, Detroit: Concerning the allergic phase of the thymic question, most pediatricians have had occasion to see patients in allergic shock after they have partaken of food to which they are extremely sensitive or after they have suddenly been exposed to an overdose of inhalants. These patients are

often in an extremely serious state, and one wonders why no one has ever described death during such an incident.

Many similarities exist between the picture of shock and that of thymic death, namely, (1) suddenness of death; (2) lack of previous symptoms and the scarcity of pathologic changes at autopsy; (3) a familial tendency; (4) the so-called thymicolymphatic diathesis, now attributed to allergy, eczema, frequent colds or lymphoid enlargement (in a follow-up study, by Anthony and myself, of children who had had roentgen treatment of the thymus [cases carefully selected on the basis of definite roentgen evidence of an enlarged thymus] the majority were found to be allergic in later years); (5) pathologic conditions noted at autopsy on patients dying from injection of animal serum and observed after thymic death; (6) true allergic asthma in its early stage, particularly in early childhood, and so-called thymic asthma, both characterized by profound shock, evidence of pulmonary edema and an ensuing pulmonary infection simulating bronchopneumonia. For this reason I believe that what has been called "thymic asthma" is actually true allergic asthma in its very beginning.

In view of this theory one may be inclined to speculate on the question, "What is the function of the thymic gland?" It is true that there must be some relation between the thymus and certain endocrine glands, particularly the adrenals, as is brought out by the work of Marine. However, anatomically the thymus is primarily a lymph gland, and as such it is involved, like other lymphoid tissue, in rendering innocuous and in eliminating from the system antigenic substances which have entered it by infection, injection, ingestion or inhalation. Other evidence (Waldbott, G. L.: *Am. J. Dis. Child.* **49**:1531, 1935) indicates that in early life lymphoid tissue plays a major part in the defense of the system against invading antigen.

I am much pleased to hear some one agree with this theory of the anaphylactic origin of so-called thymic death.

COMPARISON OF DIFFERENT METHODS IN USE FOR DETERMINATION OF SULFANILAMIDE IN TISSUES AND FLUIDS OF THE BODY. DR. JOHN J. ENGELFRIED, Ann Arbor, Mich.

During the past few years several methods have been used for the quantitative determination of sulfanilamide in blood, urine, spinal fluid and tissues of the body. Fuller (*Lancet* **1**:194, 1937), in January 1937 described a method using trichloroacetic acid for precipitation of the blood proteins, diazotizing the protein-free filtrate by adding sodium nitrite, then coupling the diazo compound with thymol in an alkaline solution to give an orange-yellow dye. The conjugated sulfanilamide was hydrolized by hydrochloric acid and then determined as sulfanilamide.

Marshall, Emerson and Cutting (*J. A. M. A.* **108**:953, 1937), in March 1937 reported that they used 96 per cent alcohol as the protein precipitant, then diazotized with sodium nitrite and coupled the resulting compound with dimethylnaphthylamine in an acid solution to give a purplish red azo compound. Later in 1937 Marshall (*Proc. Soc. Exper. Biol. & Med.* **36**:422, 1937) introduced toluenesulfonic acid as the precipitant. With this reagent it is not necessary to further acidify the filtrate before addition of the sodium nitrite. He also obtained satisfactory results using trichloroacetic acid as the precipitant, with this same azo dye. The use of a permanent standard was recommended by Proom (*Lancet* **1**:260, 1938) in January 1938.

Marshall and Litchfield (*Science* **88**:85, 1938) modified the previous methods by destroying the excess nitrous acid after diazotization with sodium nitrite and buffering the filtrate. They used sodium dihydrogen phosphate containing ammonium sulfamate. They reported that more rapid and more permanent colors are obtained and that destruction of excess nitrous acid prevents any possible destruction of the azo dye in the presence of sodium chloride and other substances.

A comparison of the various methods for the analysis of free sulfanilamide in blood shows that Marshall's original alcohol method, the trichloroacetic acid method and the p-toluenesulfonic acid method give similar results. However, in some cases slightly lower figures were obtained with the trichloroacetic acid and the p-toluenesulfonic method when the excessive nitrous acid was not destroyed.

TABLE 1.—*Comparison of Sulfanilamide Concentrations in Human Blood as Determined by Various Methods*

Method of Determination, Type of Filtrate Used	Free Sulfanilamide Concentration in Blood, Mg. Per 100 Cc.					
	Specimen 1	Specimen 2	Specimen 3	Specimen 4	Specimen 5	Specimen 6
Acidified alcoholic filtrate	10.9	6.8	5.4	4.7	2.1	1.9
Trichloroacetic acid filtrate	11.0	7.0	5.4	4.7	2.2	1.9
P-toluenesulfonic acid filtrate	11.0	...	5.2	4.8	2.2	1.8
Trichloroacetic acid filtrate with excessive nitrous acid destroyed	10.7	6.4	5.2	4.4	2.0	1.8
P-toluenesulfonic acid filtrate with excessive nitrous acid destroyed	10.8	...	5.2	4.5	2.0	1.8

TABLE 2.—*Concentration of Sulfanilamide in Blood as Determined by Various Methods*

Method of Determination, Type of Filtrate Used	Sulfanilamide Concentration in Blood, Mg. Per 100 Cc.	
	Specimen 1	Specimen 2
Acidified alcoholic filtrate	5.38	1.87
Trichloroacetic acid filtrate	5.16	1.84
P-toluenesulfonic acid filtrate	5.20	1.80
Trichloroacetic acid filtrate with excessive nitrous acid destroyed	5.37	1.90
P-toluenesulfonic acid filtrate with excessive nitrous acid destroyed	5.16	1.84

JUVENILE ASTHMA. DR. MERYL M. FENTON, Ann Arbor, Mich.

The exciting cause of asthma may or may not be protein sensitization. The cause as a rule may be determined by history, physical examination, cutaneous tests, roentgen study, the von Pirquet test, laboratory studies including nasal smears, and special tests such as the passive transfer, the autopassive transfer, the ophthalmic and the mucosal test. Asthma in childhood is usually allergic; the incidence is 1 in 200. The pathology of asthma in childhood shows a remarkable freedom from actual disease as determined bronchoscopically. Roentgen examination may show moderate sinobronchitis or glandular enlargement and markings. There are three clinical types: (1) asthmatic cough, which occurs beyond infancy and is resistant to medication; it represents the so-called preasthmatic state; (2)

asthmatic bronchitis, which occurs in infancy and in adult life, and (3) true clinical asthma, which occurs from the fourth year to puberty.

The asthmatic cough may be diagnosed by its association with a family history of allergy and by the presence of other allergic symptoms. With a bilateral family history of allergy about 75 per cent of the offspring will be allergic, two thirds giving evidence before the age of 10 years; with a unilateral family history, 50 per cent will be allergic and one third will manifest symptoms before the tenth year. The allergens derived from foci of infection usually give delayed clinical reactions; cutaneous tests and passive transfer tests may also give negative reactions. These delayed allergic reactions are responsible for many of the unsatisfactory results of cutaneous testing. During the first three years foods are the most important factors producing asthma; from 3 to 5 years, foods plus inhalants, and from 5 years to the time of puberty, inhalants, the most common being pollens, feathers, epidermal substances or animal hairs, orris root, cottonseed, kapok and silk. Infections alone as a rule do not produce asthmatic attacks.

That there may be a reflex relation between the nasal cavity, the ear canals, the mouth and throat and the bronchioles, via the vagus, has been demonstrated by Phillips.

In the study made by my co-workers and me of 50 cases of asthma in children at the University Hospital, in no case did the disease develop after the age of 10 years. The average time that elapsed between the onset of symptoms and consultation was four and four-tenths years. Our results are almost identical with those of Peshkin, who in 1934 reported a series of 100 cases of asthma in children.

Our observations demonstrate that the intradermal test is superior to the scratch test.

The treatment of asthma may be considered under three headings: (1) general hygiene, including a well balanced diet with an excess of minerals and vitamins, sufficient rest, proper humidity and temperature and avoidance of a dusty environment; (2) specific desensitization measures, with use of elimination diets, avoidance of inhalants to which the patients are sensitized, autogenous vaccines, epinephrine and ephedrine (epinephrine 1:100 inhalant has not been as successful in our hands for children as it has been reported for adults), and (3) nonspecific treatment, consisting of intravenous administration of aminophyllin, calcium salicylate and sodium salicylate and subcutaneous administration of stock vaccines. Autohemotherapy was not of great benefit in our small series. It has been difficult to determine the actual value of the various forms of therapy. Since asthma as a clinical form of allergy affects the bronchial tubes, the general body development and the personality, the importance of early diagnosis and treatment cannot be too greatly stressed. The fact that four years elapsed between the onset of symptoms and consultation demonstrates that in many cases the disease in its early stage is still being missed.

DISCUSSION

DR. J. E. BACON, Dubuque, Iowa: Do you routinely treat your patients with asthma with dust antigen whether the skin test is positive or not?

DR. MERYL M. FENTON, Ann Arbor, Mich.: If there are other antigens of the inhalant group to which the patient gives definite reactions and a suggestive history, I do not treat with dust. However, if there is no improvement with the routine measures, then I believe dust desensitization should be given a trial.

DR. JOHN SHELDON, Ann Arbor, Mich.: I believe it worth while to mention that in a case of orris sensitization there is a history of difficulty such as rhinitis or asthma while the patient is attending public gatherings, theaters and parties. The history of dust sensitization is usually confined to symptoms occurring through the winter months, when the patient is inside and the windows are closed. I have found the DeVilbiss no. 40 atomizer for use with a 1:100 solution of epinephrine hydrochloride to be of great value for the adult patient with chronic asthma when there are symptoms throughout the night in spite of the allergic routine.

DR. WILMA SACKS, Ann Arbor, Mich.: Last year I studied the value of autogenous vaccines and found them to be of superior value to the stock vaccines, especially in children. The patient is tested with the organisms of the throat culture, and a mixed vaccine is prepared, usually in two dilutions: 1,000,000 and 1,000,000,000 organisms, respectively, per cubic centimeter. Treatments are given at five day intervals.

DR. MERYL M. FENTON, Ann Arbor, Mich.: I again wish to emphasize the importance of the early diagnosis of asthma in children, before development of the complications and sequelae which are so difficult to clear up.

PREPARATION OF MOLD ANTIGENS FOR DIAGNOSTIC AND THERAPEUTIC PURPOSES. ROBERT LINDBERG, M.S., Ann Arbor, Mich.

The object of this study was (1) to develop a practical method for making antigens from strains of mold collected in the patient's environment and (2) to obtain an extract made up entirely from spores, since they are the contacting agent.

Strains of mold were isolated on Sabouraud's agar plates exposed in the home and incubated in the laboratory at 35 to 37 C. Various strains of mold were encountered, but thus far only a few genera have been studied: Penicillium, Aspergillus, Alternaria and Cladosporium as well as Monilia. Sabouraud's agar or a meat extract maltose agar was used in growing quantities of mold in 1 liter bottles, the duration of incubation being one week. The spores were harvested by washing off the surface of the culture with 35 to 50 per cent alcohol, to wet the water-repellent spores and to obtain them free from hyphae with as little agitation as possible. The concentration of alcohol was kept down to the lowest strength which would wet the spore, in order that possible denaturization of protein might be avoided. The spores were collected on filter paper in a suction funnel and dried at 37 C. for twenty-four hours. Most of the spores, but not all, were killed by alcohol. The material was composed almost completely of spores.

To extract the spores, 0.3 to 0.5 Gm. of dried material was placed in a porcelain mortar, moistened with water and frozen in a dry ice freezing mixture with methyl diethylene glycol (methyl cellosolve) at —70 C. The frozen material was ground for ten minutes with a pestle, a small amount of sterile sand being added, if necessary, to prevent caking on the walls of the mortar. The material was thawed, grinding being continued, and the entire process was repeated. After the second thawing, the mass was suspended in 50 to 70 cc. of an 0.85 per cent solution of sodium chloride, saturated with the methyl ester of p-hydroxybenzoic acid as a preservative and extracted for forty-eight hours. Filtration through paper gave a clear filtrate, easily sterilized by passage through a Berkefeld N filter. The total nitrogen content of the solution was determined, and the solution was standardized at 5 mg. of nitrogen per hundred cubic centimeters.

Concentrations of from 13 to 20 mg. of nitrogen per hundred cubic centimeters were obtained from strains of Penicillium, Aspergillus and Alternaria. In two instances determinations of Penicillium and Aspergillus revealed about 20 per cent of the total nitrogen content as protein, precipitable by trichloracetic acid.

The method as outlined permits extraction of mold antigens virtually from the spores alone, thus permitting higher concentration of the antigenic material which comes in direct contact with the mold-sensitive patient.

HYPOGLYCEMIA IN A TWO YEAR OLD CHILD WITH SUBTOTAL REMOVAL OF THE PANCREAS. DR. ROBERT SIMPSON, Battle Creek, Mich.

A 15 month old girl had attacks of weakness, stupor and convulsions from the age of 10 months. During an afternoon attack of weakness, the blood sugar value was found to be 14 mg. per hundred cubic centimeters.

Several dextrose tolerance tests were made, the following values being typical: during fasting, 35 mg.; after fifteen minutes, 43 mg.; after thirty minutes, 75 mg.; after two hours, 56 mg.; after three hours, 26 mg., and after four hours, 20 mg.

In the epinephrine test the blood sugar value during fasting was 40 mg. The value fifteen minutes after a 5 minim (0.31 cc.) injection of a solution of epinephrine hydrochloride was 75 mg.

The spinal fluid showed slightly increased pressure; the Kahn test and the colloidal gold test gave negative results. The tuberculin reaction was negative. The calcium content of the blood was 11.7 mg. and the cholesterol content 200 mg. per hundred cubic centimeters. The urine was normal. Roentgenograms of the skull, gallbladder and upper part of the intestinal tract showed normal conditions. Red cells numbered 3,800,000 and white cells 13,500, with 58 per cent lymphocytes, 40 per cent polymorphonuclears and 2 per cent eosinophils.

A high fat diet, a high protein diet, frequent feedings and administration of thyroid, various ephedrine derivatives and sedatives were tried and found ineffective. A combination high carbohydrate diet with daily injection of 2 to 3 minims (0.12 to 0.18 cc.) of a solution of epinephrine hydrochloride proved most effective.

The blood sugar value during fasting over six months varied from 14 to 47 mg. In view of the possibility of an adenoma in the pancreas, surgical therapy was advised.

On June 17, 1938, at the age of 22 months, the patient was operated on by Dr. Russell Mustard at Battle Creek. The tail, the body and half the neck of the pancreas were removed. The postoperative course was uneventful. The pancreas was normal. The microscopic examination showed no pathologic change.

On the day of the operation the blood sugar value during fasting was 41 mg. Within one hour after operation the value was 119 mg. and four hours afterward 102 mg. For two weeks after surgical intervention the blood sugar level during fasting remained at about 57 mg. Since then the value has fluctuated between 34 and 47 mg. In other words, it remains at the preoperative level.

STRYCHNINE POISONING IN A SIXTEEN MONTH OLD INFANT. DR. WILLIAM LYON, Albion, Mich.

On the morning of Dec. 6, 1937, Edward S. found a bottle which contained 40 or more "Hemroid Tablets." The label stated that the tablets were for the internal treatment of hemorrhoids, giving the dose as 1 tablet three times a day but not stating the formula or indicating the presence of any harmful ingredient. When the mother came into the room at about 10.30 a. m. she noticed purplish staining around the child's mouth and saw the empty bottle. She immediately called a physician, who came soon and, after inspecting the bottle, ordered a tablespoonful of castor oil and said that the baby would be "all right."

The oil was given, and after the child had played for a short time he was placed in bed. He slept until about 12:30 p. m., when he awakened, cried loudly and seemed in great distress. The mother noticed twitching and stiffness of the arms and legs; the child would not try to walk. The distress and twitching grew worse for about a half hour, when there was a general convulsion which lasted two or three minutes, during which the boy breathed badly but did not lose consciousness.

At this time my office was called and as I could not leave the child was brought in, arriving at 1:20 p. m. in a convulsion with extreme rigidity and some opisthotonus and crying sharply whenever he could breathe enough to do so; the condition seemed to be a typical strychnine convulsion.

Ethyl chloride was given for nearly three minutes before relaxation was attained, then 10 grains (0.65 Gm.) of chloral hydrate in 2 ounces (60 cc.) of starch water was given by rectum. With the stomach tube about 4 ounces (118 cc.) of purple material containing mucus and oil was obtained. One pint (473 cc.) of a physiologic solution of sodium chloride was given by lavage; it returned fairly clear but stained purple with a few bits of tablet that had not dissolved.

The child was quiet and breathed much better for about eight minutes, when another convulsion sharply developed with apparently complete apnea and relaxa-

tion; death occurred about a minute later, twenty minutes after the boy's arrival at the office. The temperature was normal, and the pulse rate varied from 120 to extreme rapidity.

The stomach contents and washings were given to the Department of Health, and through them the matter was presented to federal officials and to the manufacturer. The makers stated that each tablet contained about ¼ grain (0.016 Gm.) of powdered nux vomica, so that evidently the child had taken approximately 10 grains (0.65 Gm.) of the poisonous material.

My main reason for reporting this case is to emphasize the advice that the manufacturers of all drugs sold to the public should state the kind and quantity of any possibly harmful ingredients and to suggest that food and drug laws will be incomplete until such regulation is made and enforced.

PITUITARY THERAPY AND CARBOHYDRATE TOLERANCE. DR. ERNEST H. WATSON, Detroit.

The main impetus for studies of the relation between the pituitary and the pancreas came from the work of Houssay on depancreatized and pituitaritectomized

TABLE 3.—*Analysis of Iodide Content of Forty-Seven Packages of Iodized Salt Obtained on the Open Market in Michigan* *

Number of Brands	Amount of Iodide Found, %	Number of Brands	Amount of Iodide Found, %
2	0.010	10	0.020
2	0.011	4	0.021
3	0.012	3	0.022
3	0.013	2	0.023
1	0.014	1	0.024
2	0.015	0	0.025
4	0.016	1	0.026
4	0.018	1	0.035
3	0.019	1	0.050

* Claimed amount of iodide 0.020 and 0.023 per cent.

dogs. He showed that removal of the pituitary ameliorated the diabetes mellitus which followed pancreatectomy and, conversely, that implantation of pituitary grafts or injection of extract of the anterior lobe of the pituitary aggravated it.

Recently Young[7] has shown that injection of anterior pituitary extract into the intact dog causes on the third or fourth day a diabetic type of dextrose tolerance curve. If daily injections of pituitary extract are continued at the original level, the dog soon returns to normal ability to assimilate dextrose. If, however, the dose of pituitary extract injected daily is increased every few days, the dog can be rendered permanently diabetic. Dogs rendered permanently diabetic by continued injection of increasing doses of pituitary extract show hyalinization of the islets which is apparently the end result.

There have been occasional reports of patients showing glycosuria and decreased dextrose tolerance following injection of anterior pituitary extract containing the growth hormone and of the anterior pituitary-like gonadotropic factor from the urine of pregnant women. The percentage of patients reacting in this manner is not great.

Of a group of 10 patients, it was noted in 1, who was carefully studied by Dr. J. A. Johnston, of Henry Ford Hospital, that injection of the extract containing

7. Young, F. G.: Proc. Roy. Soc. Med. **31**:1305, 1938.

the growth hormone caused a definite change in the dextrose tolerance curve and in the phosphorus curve toward the diabetic type. Discontinuing administration of the extract resulted in a return to normal of the dextrose tolerance and in sugar-free urine.

This brief review is presented with the hope that it may stimulate continued observation of dextrose metabolism in patients under treatment with endocrine preparations, especially pituitary extracts. Until all the facts are known, it cannot be assumed that the use of pituitary extracts carries any serious danger of impairment of carbohydrate metabolism through its effects on the pancreas.

DISTRIBUTION AND CONTENT OF IODIDE IN IODIZED SALT OBTAINED ON THE OPEN MARKET THROUGHOUT MICHIGAN. DR. D. MURRAY COWIE and DR. JOHN J. ENGELFRIED, Ann Arbor, Mich.

We have made a survey of the iodide contained in the iodized salt obtained on the market throughout the northern and southern peninsulas of Michigan. The method of analysis was method 2 of the Department of Agriculture, Washington, D. C., which employs chloroform, sulfuric acid and hydrogen peroxide as the liberating and collecting mediums. Of 47 packages of salt examined, representing 29 different brands, but evidently not all different salts, the iodide content varied from 0.010 to 0.050 per cent. The amount claimed on the package was either 0.020 or 0.023 per cent. Table 3 gives the number of packages containing the varying amounts.

This shows that no salt contained less than 0.010 per cent of iodide, the amount originally recommended by the Iodized Salt Committee of the Pediatric Section of the Michigan State Medical Society, as the amount that should be sufficient to prevent the development of endemic goiter; in 2 packages the amount was distinctly in excess. The analysis further shows the wisdom of selecting a higher value than 0.010 per cent as the required amount for the marketed product. It was believed, for several reasons, that there would be a tendency to a lower content. This is shown in 44 per cent of the packages. The analysis suggests the advisability of routine inspection of the marketed product. Observations in the complete report consider the explanation for the variation in iodide, its distribution throughout the package and the type of package, as well as the amount of iodide in the contents of the package itself.

News and Comment

GENERAL

Meeting of Pediatric Section.—At the forthcoming annual session of the American Medical Association, to be held in New York City June 10 to 14, 1940, the Section on Pediatrics will have its headquarters and hold its meetings at the Waldorf-Astoria. The banquet of the section, on June 13, will also be held at that hotel.

SOCIETY NEWS

American Orthopsychiatric Association.—The seventeenth annual meeting of the American Orthopsychiatric Association, an organization for the study and treatment of behavior and its disorders, will be held at the Hotel Statler, Boston, on Feb. 22 to 24, 1940. Dr. Norvelle C. LaMar, 149 East Seventy-Third Street, New York, is secretary.

DEATHS

Dr. Isidor H. Tumpeer, an attending pediatrician at Michael Reese Hospital, and head of the department of pediatrics at Post Graduate Hospital, Chicago, died on November 29 at the age of 46 years.

CORRECTION

In the article entitled "Immunologic Properties of Scarlatina Convalescent Serum" by Drs. Elizabeth Moore and William Thalhimer, in the November issue (AM. J. DIS. CHILD. **58**:1039, 1939), in the fourth line from the top on page 1043, 49 should read 42; 73, 63; 29, 25, and 43, 37.

Notices

CUMULATED INDEX OF THE AMERICAN JOURNAL OF DISEASES OF CHILDREN

Requests have been received for a twenty year index of the AMERICAN JOURNAL OF DISEASES OF CHILDREN. Before serious consideration is given to the production of a cumulated index, it is desirable to know whether the demand for it would be sufficient to warrant its sale at not to exceed $6 per copy; that is, whether one thousand copies could be sold. It will be appreciated if those who are interested in such an index will fill out the accompanying blank and send it to the Managing Editor at the publication office, 535 North Dearborn Street, Chicago.

I SHOULD BE WILLING TO SUBSCRIBE TO A CUMULATED INDEX OF THE AMERICAN JOURNAL OF DISEASES OF CHILDREN AT $6.00.

NAME .....

ADDRESS .....

.....

DATE.....

Book Reviews

Die Pflege des gesunden und kranken Kindes. By Werner Catel, M.D. Price, paper 18 marks, bound 19.60 marks. Pp. 764, with 423 illustrations. Leipzig: Georg Thieme, 1939.

This book is compiled with the usual German thoroughness. It contains a wealth of material. The illustrations, partly colored, are excellent and portray much that is of a great deal of value in the practice of pediatrics.

The first section, which is 203 pages long, consists of basic facts, extensive treatises on the anatomy, physiology and metabolism of the child, together with exhaustive considerations of genetics and hygiene. The second section takes up the care of the well child and contains chapters on health and disease in the woman and mother; development and nourishment of the healthy baby; care of the healthy baby; development, nourishment and care of the sound child and the school child, and the peculiarities and care of the premature child. There is a chapter on the psychology of the child and one on play. It is interesting that in this section more than twenty-five pages are taken up with descriptions of games and playthings.

The third section has to do with the care of the sick child. The first part of this takes up the care of the infant and older child, next comes a chapter on infectious diseases, and then one on the bases for surgical treatment. The latter is replete with pictures of surgical instruments. There follows a chapter on the care of the sick infant and the sick child, one on milk and diet kitchens, one on drugs, one on disinfectants, one on electrical apparatus to be used and one on accidents. Many of the measures advocated throughout this book are not in use in this country, and the prescriptions for infant feeding contain no mention, so far as the reviewer could find, of Marriott's lactic acid milk.

On the whole the volume is a useful one and should prove of great value as a reference work.

Co-authors with Catel are R. Büttner, of Dresden; F. Claussen, of Cologne; and E. Hässler, I. Kloss, R. Schröder, R. Sievers and I. Zenker, of Leipzig.

Epidemic Encephalitis: Etiology, Epidemiology, Treatment. Third Report by the Matheson Commission. Price, $3. Pp. 493, with no illustrations. New York: Columbia University Press, 1939.

This volume summarizes the advances made in the knowledge of epidemic encephalitis from 1930 to 1937. Of particular value is the discussion of the St. Louis type of encephalitis which occurred in 1933 and the Japanese B type which occurred in 1934-1935. There are also a short discussion of encephalitis caused in man by the viruses of Eastern and Western equine encephalomyelitis and discussions of other types of encephalitis, such as the postvaccinal and the hemorrhagic form.

More than half the volume is devoted to the bibliography of encephalitis and allied diseases from 1930 to 1937. The publication contains the most exhaustive study and the most complete bibliography of encephalitis to be found in any language.

Directory of Pediatric Societies*

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof. S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

President: Prof. Luigi Spolverini, Policlinico Lungo Tevere Melini 24, Rome, Italy.
Secretary: Professor Vitetti, Rome, Italy.
The meeting is postponed to an indefinite date.

American Committee:
Chairman: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
General Secretary: Dr. Kenneth D. Blackfan, 300 Longwood Ave., Boston.
Assistant Secretary-Treasurer: Dr. Charles F. McKhann, 300 Longwood Ave., Boston.

Canadian Committee:
Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

British Paediatric Association

President: Prof. Charles McNeil, 44 Heriot Row, Edinburgh, Scotland.
Secretary: Dr. Arthur G. Maitland Jones, 31A Weymouth St., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. E. Gjørup, Dronning Louises Børnehospital, Copenhagen.

Paediatrický Spolok na Slovensku

President: Dr. A. J. C. Chura, Lazaretská 11, Bratislava.
Secretary: Dr. P. Rados, Lazaretská 6, Bratislava.
Place: Pediatric Clinic, University Bratislava. Time: Six times a year.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. E. A. Cockayne, 98 Harley St., London, W. 1, England.
Secretary: Dr. R. Lightwood, 86 Brook St., London, W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. S. Rosenbaum, 26 Bialikstr., Tel Aviv.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Sociedad Cubana de Pediatría

President: Dr. Clemente Inclan Costa, Manrique 48, Habana.
Secretary: Dr. Serafin Falcon Lopez, Calle B No. 356, Vedado, Habana.
Place: Cátedra de Clinica Infantil, Hospital Mercedes, Habana. **Time:** Last Wednesday of every month.

Sociedad Mexicana de Pediatria

President: Dr. Jorge Muñoz Turnbull, Av. Alvaro Obregón 186, Mexico, D. F.
Secretary: Dr. Fernando López Clares, Medellín 191, Mexico, D. F.

Société de Pédiatrie de Paris

President: Dr. B. Weill-Hallé, 49 Avenue Raymond Poincaré, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue du Pré aux Clercs, Paris, France.
Place: Hôpital des Enfants Malades, 49 Rue de Sèvres. **Time:** 4:30 p. m., third Thursday of every month.

NATIONAL

American Medical Association, Scientific Assembly, Section on Pediatrics

Chairman: Dr. Albert D. Kaiser, 16 N. Goodman St., Rochester, N. Y.
Secretary: Dr. Hugh L. Dwyer, 315 Alameda Rd., Kansas City, Mo.
Place: New York. **Time:** June 10-14, 1940.

American Academy of Pediatrics

President: Dr. H. F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.
Place: Hotel Peabody, Memphis, Tenn. **Time:** Nov. 18-20, 1940.
Region I. Place: Mayflower Hotel, Washington, D. C. **Time:** April 4-6, 1940.
Region II. Place: Edgewater Gulf Hotel, Edgewater Park, Miss. **Time:** March 22-23, 1940.
Region IV. Place: Seattle, Wash. **Time:** May 30-June 1, 1940.

American Hospital Association, Children's Hospital Section

Chairman: Miss Mabel Binner, Children's Memorial Hospital, Chicago.
Secretary: Winifred Culbertson, Children's Convalescent Hospital, Cincinnati.

American Pediatric Society

President: Dr. Herbert B. Wilcox, 39 E. 75th St., New York.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid St., St. Louis.

Canadian Society for the Study of Diseases of Children

President: Dr. L. M. Lindsay, 815 Medical Arts Bldg., Montreal.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, Hospital for Sick Children, Toronto.

Society for Pediatric Research

President: Dr. Daniel C. Darrow, 330 Cedar St., New Haven, Conn.
Secretary: Dr.Mitchell I. Rubin, 1740 Bainbridge St., Philadelphia.

SECTIONAL

Intermountain Pediatric Society

President: Dr. Eugene Smith, 385–24th St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City.
Place: Salt Lake City General Hospital. **Time:** First Thursday of each month, 8 p. m.

New England Pediatric Society

President: Dr. R. Cannon Eley, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. James Marvin Baty, 319 Longwood Ave., Boston.
Place: Boston Medical Library. **Time:** Four meetings a year, occurring from September to May.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. Frederick Fischer, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. C. L. Lyons, Paulsen Medical-Dental Bldg., Spokane, Wash.

ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. John Schoonover, Republic Bldg., Denver.
Secretary: Dr. F. Craig Johnson, 1850 Gilpin St., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 7:30 p. m.

SOUTHERN MEDICAL ASSOCIATION, SECTION OF PEDIATRICS

Chairman: Dr. Hughes Kennedy Jr., Highland Plaza Apts., Birmingham, Ala.
Secretary: Dr. Warren W. Quillian, 227 Aragon Ave., Coral Gables, Fla.

STATE

ALABAMA PEDIATRIC SOCIETY

President: Dr. Stewart H. Welch, 1117 S. 20th St., Birmingham.
Secretary-Treasurer: Dr. William R. Britton, 4 Catoma St., Montgomery.

ARKANSAS STATE PEDIATRIC ASSOCIATION

Chairman: Dr. G. D. Murphy, 112 Peach St., El Dorado.
Secretary: Dr. Madeline M. Melson, 909 Main St., Little Rock.

CALIFORNIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Dr. Harold K. Faber, 2341 Clay St., San Francisco.
Secretary: Dr. Hartzel H. Ray, 23-2d Ave., San Mateo.

FLORIDA STATE PEDIATRIC SOCIETY

President: Dr. Warren W. Quillian, 227 Aragon Ave., Coral Gables.
Secretary: Dr. George N. Leonard, 605 Lincoln Rd., Miami Beach.
Place: Concurrent with state association meeting at time of convention.

GEORGIA PEDIATRIC SOCIETY

President: Dr. L. H. Muse, 384 Peachtree St. N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
Scientific Meeting: Place: Atlanta. Time: December 1939. Business Meeting: Place: Savannah. Time: April 1940.

HEZEKIAH BEARDSLEY PEDIATRIC CLUB OF CONNECTICUT

President: Dr. Oliver L. Stringfield, 1416 Bedford St., Stamford.
Secretary: Dr. Joseph L. Hetzel, 103 N. Main St., Waterbury.
Time: Three meetings a year.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Dr. H. William Elghammer, 5307 Hyde Park Blvd., Chicago.
Secretary: Dr. Bert I. Beverly, 8 S. Michigan Ave., Chicago.
Place: Peoria. Time: May 22, 1940.

INDIANA STATE PEDIATRIC SOCIETY

President: Dr. O. N. Torian, 23 E. Ohio St., Indianapolis.
Secretary-Treasurer: Dr. Mathew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

IOWA PEDIATRIC SOCIETY

President: Dr. Morgan J. Foster, 837 Higley Bldg., Cedar Rapids.
Secretary-Treasurer: Dr. Arnold M. Smythe, 406-6th Ave., Des Moines.

Louisiana State Pediatric Society

President: Dr. Phillip C. De Verges, Audubon Bldg., New Orleans.
Secretary-Treasurer: Dr. W. C. Rivenbark, 220 Physicians and Surgeons Bldg., New Orleans.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. Paul W. Beaven, 26 S. Goodman St., Rochester.
Secretary: Dr. Leslie O. Ashton, 850 Park Ave., New York.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. John M. Higgins, 600 S. Wilbur Ave., Sayre.
Secretary: Dr. John D. Sturgeon Jr., 22 N. Gallatin Ave., Uniontown.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Allan L. Richardson, 3001 W. Grand Blvd., Detroit.
Secretary: Dr. Ward L. Chadwick, 524 Medical Arts Bldg., Grand Rapids.

Mississippi State Pediatric Society

President: Dr. Guy C. Jarratt, 920 Crawford St., Vicksburg.
Secretary-Treasurer: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Place: To be designated by society. Time: Yearly.

Nebraska Pediatric Society

President: Dr. Herman M. Jahr, 1120 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. Elmer W. Bantin, 440 Aquila Court Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. Marion Y. Keith, 369 N. Elm St., Greensboro.
Secretary: Dr. G. W. Kutscher Jr., 176 Woodfin St., Asheville.

Oklahoma State Pediatric Society

President: Dr. A. L. Salomon, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. Ben H. Nicholson, 301 W. 12th St., Oklahoma City.

South Carolina Pediatric Society

President: Dr. D. Lesesne Smith Jr., 187 Oakland Ave., Spartanburg.
Secretary-Treasurer: Dr. William Weston Jr., 1428 Lady St., Columbia.

Tennessee State Pediatric Society

President: Dr. William E. Van Order, 546 McCallie Ave., Chattanooga.
Secretary: Dr. Kinsey M. Buck, 1193 Madison Ave., Memphis.

Texas Pediatric Society

President: T. A. Tumbleson, 2100 North St., Beaumont.
Secretary-Treasurer: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Place: Dallas. Time: May 15, 1940.

Virginia Pediatric Society

President: Dr. W. Ambrose McGee, 616 W. Grace St., Richmond.
Secretary: Dr. John M. Bishop, Medical Arts Bldg., Roanoke.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. J. L. Blanton, 207 Fairmont Ave., Fairmont.
Secretary: Dr. Theresa O. Snaith, 450 Center Ave., Weston.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. N. C. Wetzel, 2103 Adelbert Rd., Cleveland.
Secretary: Dr. Fred Rittenger, 10515 Carnegie Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. T. B. Verner, 170 St. George St., Toronto, Canada.
Secretary: Dr. D. V. Hutton, 170 St. George St., Toronto, Canada.

Bronx Pediatric Society

President: Dr. Samuel T. Levethan, 254 E. 184th St., Grand Concourse, New York.
Secretary: Dr. David Greene, 910 Grand Concourse, New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

Brooklyn Academy of Pediatrics

President: Dr. Benjamin Kramer, 6 Pierrepont St., Brooklyn.
Secretary: Dr. John A. Monfort, 861 Carroll St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Fourth Wednesday of each month, except May, June, July, August and September.

Central New York Pediatric Club

President: Dr. Frank van der Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Third Thursday of April and September.

Chicago Pediatric Society

President: Dr. Gerard N. Krost, 2376 E. 71st St., Chicago.
Secretary: Dr. George F. Munns, 723 Elm St., Winnetka, Ill.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Dallas Pediatric Society

President: Dr. Harold Nesbit, 1719 Pacific Ave., Dallas, Texas.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmont Ave., Dallas, Texas.
Place: Bradford Baby Hospital. Time: 1 p. m., second and fourth Saturdays of each month.

Detroit Pediatric Society

President: Dr. William O'Donnell, 1553 Woodward Ave., Detroit.
Secretary: Dr. Robert Mason, 5140–2d Blvd., Detroit.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month from October to June, inclusive.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. L. H. Goldsmith, 384 Peachtree St. N. E., Atlanta.
Secretary: Dr. James P. Hanner, 384 Peachtree St. N. E., Atlanta.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Houston Pediatric Society

President: Dr. James H. Park Jr., 3717 Main St., Houston, Texas.
Secretary-Treasurer: Dr. Thomas J. Donovan, 2418 Travis St., Houston, Texas.
Place: Houston Club. Time: Third Monday of each month.

Kansas City (Missouri) Pediatric Society

President: Dr. Urban Busiek, 200 E. Pershing St., Springfield, Mo.
Secretary: Dr. Fred Speer, 2700 Parallel Ave., Kansas City, Kan.
Place: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

President: Dr. H. R. Cooder, 3875 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. Paul M. Hamilton, 248 E. Main St., Alhambra, Calif.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd.
Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Benjamin Stoloff, 1149 Eastern Parkway, Brooklyn.
Secretary: Dr. Harry Apfel, 988 Park Pl., Brooklyn, N. Y.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., second Monday of each month, October to March, inclusive.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. James Dobbins, 42-04 Ditmars Blvd., Long Island City, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144–38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry H. Donnally, 1612 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Harry A. Spigel, 2647 Connecticut Ave. N. W., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of October, January, February, March and April.

Memphis Pediatric Society

President: Dr. Barton Etter, 376 S. Bellevue St., Memphis, Tenn.
Secretary-Treasurer: Dr. Harry Jacobson, 1193 Madison Ave., Memphis, Tenn.
Place: John Gaston Hospital. Time: Quarterly.

Milwaukee Pediatric Society

President: Dr. Helen Jane Zillmer, 781 N. Jefferson St., Milwaukee.
Secretary-Treasurer: Dr. Samuel E. Kohn, 425 E. Wisconsin Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Rustin McIntosh, Babies Hospital, New York.
Secretary: Dr. Leslie O. Ashton, 850 Park Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northwestern Pediatric Society

President: Dr. Robert Rosenthal, 437 Lowry Medical Arts Bldg., St. Paul.
Secretary-Treasurer: Dr. R. E. Nutting, 205 W. 2d St., Duluth, Minn.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Oklahoma City Pediatric Society

President: Dr. A. L. Salomon, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 1200 N. Walker St., Oklahoma City.
Time: Every month, except June, July and August.

Philadelphia Pediatric Society

President: Dr. Samuel Goldberg, 4546 N. Broad St., Philadelphia.
Secretary: Dr. Aims C. McGuinness, Children's Hospital, 1740 Bainbridge St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to April, inclusive.

Pittsburgh Pediatric Society

President: Dr. James LeRoy Foster, Highland Bldg., Pittsburgh.
Secretary-Treasurer: Dr. E. R. McClusky, 3200–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate month from October to June, inclusive.

Richmond Pediatric Society

President: Dr. Emily Gardner, 1100 W. Franklin St., Richmond, Va.
Secretary-Treasurer: Dr. T. Stanley Meade, 913 Floyd Ave., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: 8 p. m., first Thursday of each month, except July, August and September.

Rochester Pediatric Society

President: Dr. David Wolin, 16 N. Goodman St., Rochester, N. Y.
Secretary: Dr. H. C. Soule, 16 N. Goodman St., Rochester, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Second Friday of each month.

St. Louis Pediatric Society

President: Dr. Max Deutch, 634 N. Grand Blvd., St. Louis.
Secretary-Treasurer: Dr. Peter G. Danis, 634 N. Grand Blvd., St. Louis.
Place: St. Louis Medical Society Bldg. Time: Third Friday of each month from September to June.

Seattle Pediatric Society

President: Dr. Jay I. Durand, 1305–4th Ave., Seattle.
Secretary: Dr. Frederick B. Joy, 1215–4th Ave., Seattle.
Place: College Club. Time: Third Friday of each month, 6:30 p. m.

Southwestern Pediatric Society

President: Dr. E. Earl Moody, 829 S. Alvarado Ave., Los Angeles.
Secretary: Dr. Norman Nixon, 1136 W. 6th St., Los Angeles.
Place: Jonathan Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Clement Smith, 1101 Beacon St., Brookline, Mass.
Secretary: Dr. D. Murray Cowie, University of Michigan, Ann Arbor, Mich.

Westchester County Medical Society, Pediatric Section (New York)

President: Dr. Norman Survis, 650 Main St., New Rochelle.
Secretary-Treasurer: Dr. Elvira Ostlund, 131 Purchase St., Rye.
Place: Grasslands Hospital, Valhalla, N. Y. Time: Third Thursday in October, December, February and April.

INDEX TO VOLUME 58

The asterisk (*) preceding the page number indicates an original article. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Book Reviews, Obituaries and Society Transactions are indexed under the letters B, O and S, respectively.

Abbott, O. D.: Iron deficiency anemia in children, *811

Abdomen: See also Gastrointestinal Tract; Pelvis
- congenital absence of abdominal musculature: associated changes in genitourinary tract and in spinal cord, *339
- danger of primary abdominal tuberculosis in children, 618
- difficulties in abdominal diagnosis in children, 1348
- left upper abdominal pain on effort in juvenile rheumatic disease; new symptom of low grade active phase, 865
- urgent early diagnoses in life-threatening diseases of abdominal organs in childhood, 423

Abnormalities and Deformities: See also Achondroplasia; Acrocephaly; Cripples; Hypertrophy; Monsters; Twins; and under names of diseases, organs and regions, as Appendix, abnormalities; Brain, abnormalities; Colon, abnormalities; Fingers and Toes, abnormalities; Heart, abnormalities; Lungs, absence of; Spine, abnormalities; Wrist, deformities; etc.
- congenital deformities of skeleton, 1101
- plastic surgery in children; medical and psychologic aspects of deformity, 441
- surgical treatment of malformations and its relation to law regarding prevention of hereditary diseases, 1134

Abscess: See also under names of organs and regions, as Brain, abscess; Liver; Lungs, abscess; etc.
- retropharyngeal, 623
- retropharyngeal, severe hemorrhage secondary to, *1256
- retropharyngeal, with fistulization into external auditory canal, 874
- tonsillectomy in presence of acute peritonsillar abscess, 874

Acacia, reaction of edematous patient to transfusion with serum or with acacia, 915

Accommodation: See Eyes, accommodation and refraction

Acetarsone, eruptive fever (dengue, measles, German measles?) in reality complex toxic drug dermatitis (pentavalent arsenic and sulfanilamide derivative), 650
- stovarsol intoxication, 1136

Achalasia: See under Esophagus

Achondroplasia, role of advancing maternal age in causing achondroplasia, *994

Acid, Amino: See Aminoacids
- Ascorbic: See Scurvy; Vitamins, C
- Cevitamic: See Scurvy; Vitamins, C
- fatty: rat acrodynia and essential fatty acids, 1347
- lactic, formation by muscles of scorbutic guinea pigs, 1343
- lactic, metabolism in acute rheumatic fever, 1138
- Mandelic: See under Urinary Tract
- Nicotinic: See Pellagra
- Organic: See under Urinary Tract
- Phosphoric: See Phosphorus and Phosphorus Compounds

Acid-Base Equilibrium: See also Acidosis
- effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
- role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072

Acidosis, certain aspects of ketosis and acidosis, 598
- in infants of Bogota, Colombia, 851
- late rickets with glycosuria and organic acid acidosis (Fanconi type), 673
- role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072

Acridine Dyes, blackwater fever following administration of "atebrin," 1318

Acrocephaly, craniofacial dysostosis, 857
- oxycephaly and alterations of optic nerve, 402
- oxycephaly: new operation and its results (preliminary report), 400
- true oxycephaly, 402

Acrodynia, 898
- malignant type, 897
- pink disease, 896
- rat acrodynia and essential fatty acids, 1347

Adams-Stokes' Disease: See Heart, block

Adenitis: See Lymph Nodes

Adenoids, acute anterior poliomyelitis following tonsillectomy and adenoidectomy with reference to bulbar form, 1105
- biologic significance of tonsils and adenoids and other external lymphoid masses, 1113
- local anesthesia in children, 1134

Adenosarcoma: See under Sarcoma

Adie Syndrome: See under Reflex

Adipose Tissue: See Fat; Obesity

Adiposogenital Syndrome: See Laurence-Biedl Syndrome

Adolescence, behavior problems of, *168
- gynecologic problems of adolescent girl, *162
- induction of premature puberty with androgenic substance, 889
- noninfectious elevation in temperature among adolescents, 1349
- precocious sexual development, 1323
- pseudosexual precocity; adrenal cortical syndrome in preadolescent girls, 1126
- some hysterical syndromes at puberty and at prepubertal periods, 638
- treatment of acute osteomyelitis in, 1344

Adoption and children of syphilitic mothers, 198

Adrenal Preparations, certain therapeutic effects of whole suprarenal gland by mouth, 888
- experimental studies on treatment of malarial splenomegalies by method of Ascoli, 1108
- insensible perspiration in children; influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71

Adrenalectomy: See under Adrenals

Adrenalin, Therapy: See Asthma

Adrenals; adrenal virilism, 643
- capsular hemorrhages in newborn infants, 186
- effect of thyroidectomy and of adrenalectomy on thymus of castrated white rat, 1342
- infant Hercules type of adrenogenital syndrome, 1341
- neuroblastoma of left adrenal with intrathoracic metastases, 432

Adrenals—Continued
pseudosexual precocity; adrenal cortical syndrome in preadolescent girls, 1126
tumors in early childhood with report of 2 cases, 890
Agar: See under Dyspepsia
Age, effect on blood pressure in infants and children, *113
experimental scurvy of guinea pig, 603
order of epiphysial union in Bengalee girls, 644
role of advancing maternal age in causing achondroplasia, *994
Agranulocytosis: See Granulocytopenia
Ahmann, C. F.: Iron deficiency anemia in children, *811
Air, atmosphere of reduced nitrogen with respiratory obstruction, 1351
contamination and air sterilization, *1023
Air Passages: See Respiratory Tract
Albumin: See also under Blood, proteins; Cerebrospinal Fluid; Protein
residual nitrogen, total serum protein and albumin-globulin quotient in dyspeptic toxicosis of infants, 1334
Alcohol, effect on hunger sense, 627
Therapy: See Lungs, abscess
Aldrich, C. A.: Concentrated human blood serum as diuretic in nephrosis; further observations, 914
Alexander, H. E.: Response to antiserums in meningococcic infections of human beings and mice; comparative study, *746
Alimentary Intoxication: See under Digestive Tract; Toxemia
Tract: See Digestive Tract
Alkali Therapy: See Nephritis
Allen, E.: Gynecologic problems of adolescent girl, *162
Allergy: See Anaphylaxis and Allergy; Asthma; Food, allergy; Hay Fever; etc.
Alum: See under Diphtheria; Poliomyelitis
Amblyopia: See Blindness
Amebiasis, amebic dysentery in childhood, 1320
Amentia: See Feeblemindedness; Idiocy; etc.
American Medical Association, meeting of Section on Pediatrics, 1371
Aminoacids: See also Nitrogen; Protein
comparative study of metabolism of certain aminoacids with reference to respiratory exchange and heat production, 848
Therapy: See Dystrophy, muscular
Aminopyrine, Therapy: See Rheumatic Fever
Ammonia and Ammonium Compounds, role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Amnion, dropsy of; teratoma of neck of fetus with acute hydramnios, 1308
Amyotonia: See Myatonia
Anaphylaxis and Allergy: See also Asthma; Food, allergy; Hay Fever; Tuberculosis, allergy; etc.
Alimentary: See Food, allergy
allergic purpura, 221
allergy in childhood, 221
allergy in childhood; its onset and natural progress, 895
allergy in childhood; modes of its acquisition, 647
allergy in childhood; prophylaxis, 647
allergy in infants, 440
allergy in infants; significance of first allergic manifestations, 438
certain therapeutic effects of whole suprarenal gland by mouth, 888
experimental asthma, *699
experimental studies on congenital allergy, 898
importance of sensitization mechanism in clinical phenomenon of allergy; possible cause and prevention, *581
Anaphylaxis and Allergy—Continued
influence of vitamin B_1 and vitamin C on anaphylactic shock, 855
inverse anaphylaxis in man; problem of hypersensitivity in man, 650
meningeal reactions to serum, 1106
nature of allergic and nonallergic lobar pneumonias, 208
prophylaxis in allergy, 1345
role of allergy in atelectasis in children, *237
serum disease, 898
thymus and thymicolymphatic constitution; anaphylactic change, 1360
treatment of antiserum to remove serum-sickness-causing factor, 1346
Anasarca: See Edema
Andersen, D. H.: Congenital leukemia, *1268
Anderson, M.: Rate of appearance of ossification centers from birth to age of 5 years, *949
Anderson, N. A.: Causes of prematurity; review of literature, *586
Androgens, daily excretion of urinary androgens in normal children, 1127
effect of prolonged testosterone propionate administration on immature and adult female rat, 1126
excretion and fate of, 641
gynecomastia, 251
induction of premature puberty with androgenic substance, 889
inhibition of lactation during puerperium by testosterone proprionate, 641
progesterone is androgenic, 1127
Anemia, Agranulocytic: See Granulocytopenia
circulatory function in anemias of children; roentgenographic measurement of cardiac size, *250
Cooley's: See Pseudoleukemia, infantile
copper content of milk in anemic infants, 181
Erythroblastic: See Pseudoleukemia, infantile
hemolytic, acute; Lederer type, *349
hemolytic, congenital, with normal fragility of red blood cells, 403
hemolytic, in newborn infant, 872
hemolytic, of childhood, 621
hemolytic, severe, due to sulfanilamide, 1111
influence of cobalt on iron transportation in blood, 671
iron deficiency anemia in children, *811
nutritional, in industrial district, 871
nutritional, new carbohydrate for prevention in infants; preliminary report, *45
of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
role of copper in iron-deficiency anemia in infancy, 620
scarlet fever heart, 1316
splenic; Gaucher's disease, 873
syndrome from ascariasis, 193
vegetative regulation of red blood cells in, 1112
Anencephaly: See Monsters
Anesthesia, certain aspects of ketosis and acidosis, 598
complementing activity and ascorbic acid content of guinea pig serums following ether anesthesia, 601
cyclopropane, effect on blood pressure, *115
local, in children, 1134
Aneurysm, cardiac; congenital "aneurysmal" dilatation of left auricle, 870
of cubital artery in infant of 9 months, 1326
Angina: See also Throat; Tonsils
Agranulocytic: See Granulocytopenia
Vincent's; fusospirillar stomatitis, 1117
Angioma, hemangioma of vertebral column, 436
treatment of lymphangioma by sclerosing injections of sodium citrate, 218
Aniridia: See Iris, absence of
Anomalies: See Abnormalities and Deformities; and under names of organs and regions

Anorexia: See Appetite
Anosmia: See Smell
Anoxemia: See under Oxygen
Anspach, W. E.: Familial cleidocranial dysostosis (cleidal dysostosis); preosseous and dentinal dystrophy, *786
Hyperparathyroidism in children; report of 2 cases, *540
Anthelmintics: See Ascariasis; Intestines, parasites; etc.
Antibodies: See Antigens and Antibodies
Antigens and Antibodies: See also Immunity; Lipoids; and under specific antigens and reactions, as Staphylococci; Whooping Cough; etc.
effect of hyperimmune human serum (lyophile) on humoral antibody titer in pertussis, *1234
human serum response to gonadotropic hormone (pregnancy urine extract), 1127
preparation of mold antigens for diagnostic and therapeutic purposes, 1367
Antiserum: See Diphtheria, antiserum; etc.
Antithrombin: See Blood, coagulation
Antitoxin: See under Tetanus; etc.
Antrum: See also Sinuses, Nasal
otologic relations to dysentery in infancy, 1319
paranasal sinuses in children, 419
Antuitrin G: See Pituitary Body
Antuitrin S: See Hormones, sex
Aorta, coarctation; report of case, 417
Aphthae: See Stomatitis
Apoplexy: See Brain, hemorrhage
Apparatus, experimental asthma, *699
Appendicitis: See also Appendix
difficulties in abdominal diagnosis in children, 1348
from Oxyuris infestation, 881
in childhood, 423
peritonitis due to ruptured acute appendicitis in children; influence of delay on operative mortality, 1332
recurrent, 629
Appendix, congenital anomaly of valve of Bauhin and of appendix as cause of peritonitis in newly born, 1312
hernia of vermiform appendix, 422
Appetite; anorexia, 878
Apples, effect of apples and cranberries on calcium retention, 1093
Arachnodactylia, 1308
in 2 siblings, 1309
masked dolichostenomelia, 1101
Arachnoiditis of optic chiasm cured by encephalographic examination, 1336
Arakawa Reaction: See Oxydase and Peroxydase
Arms: See Extremities; Forearm; etc.
Arrhythmia: See also Heart, block
bigeminal pulse, 1327
extrasystolic, in scarlet fever, 1317
Arsphenamines, serous apoplexy in children in course of treatment with arsenobenzenes, 631
serous apoplexy in children; 2 cases, 631
Therapy: See Syphilis
Arteries: See also Aneurysm; Aorta; Arteriosclerosis; Blood, pressure; Embolism; Thrombosis; etc.
arterial calcification and subcutaneous calcinosis; case shown in January 1936, 896
Arthritis: See also Rheumatism; and under names of joints
atlantoaxial subluxation due to postanginal arthritis; reduction, 219
cytologic studies on rheumatic fever, 191
deformans; hemolytic streptococcus in etiology of rheumatic fever and rheumatoid arthritis, 192
deformans; new line of treatment (cysteine) in certain type of arthritis; preliminary communication, 1129
Arthritis—Continued
deformans; ocular complications in rheumatism, 1132
primary chronic infectious arthritis, 645
Ascariasis, anemia syndrome from, 193
clinical, in children, 411
epidemiology of Ascaris lumbricoides in urban population, 195
meningeal symptoms caused by Ascaris, 1321
Ascorbic Acid: See Scurvy; Vitamins, C
Ash, R.: Diagnosis of congenital cardiac defects in infancy; study of 32 cases with necropsies, *8
Asphyxia: See also under Respiration
atmosphere of reduced nitrogen with respiratory obstruction, 1351
neonatorum; relative value of pure oxygen and of carbon dioxide mixtures in experimental resuscitation, 858
Aspirin: See under Salicyl Compounds
Asthma: See also Anaphylaxis and Allergy
effectiveness of intravenous hypertonic sucrose and adrenalin in treatment of status asthmaticus, 221
experimental, *699
in children treated with autogenous (bronchoscopic) vaccine, *768
interstitial emphysema in asthmatic patient, 1116
juvenile, 1365
role of allergy in atelectasis in children, *237
thymic, 1128
Atabrine, Therapy: See Malaria
Ataxia, Friedreich's: See Ataxia, hereditary
hereditary cerebellar ataxia, 883
syndrome characterized by progressive cerebellar ataxia with feeblemindedness, 632
Atebrin: See Acridine Dyes
Atelectasis: See Lungs, collapse
Athetosis and chorea in childhood, 1119
following acute specific fevers, 193
Atlas and Axis, atlantoaxial subluxation due to postanginal arthritis; reduction, 219
Atmosphere: See Air
Atrophy: See also under names of organs and regions, as Bones; etc.
Infantile: See Celiac Disease; Infants, nutrition
muscle dystrophies and atrophies occurring in childhood from orthopedic aspect, 441
Muscular: See also Dystrophy, muscular
muscular atrophies and dystrophies in childhood, 434
Atropine, insensible perspiration in children: influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71
Auger, W. J.: New method of obtaining sputum from infants and children thought to have pneumococci in nasopharynx, 1139
Auricular Fibrillation: See Arrythmia
Avitaminosis: See under Deficiency Diseases; Vitamins; and under names of deficiency diseases, as Beriberi; Scurvy; etc.

B C G Vaccine: See under Tuberculosis
Bacal, H. L.: In vitro experiments on effect of added blood serum and blood plasma on sedimentation rate, 1137
Bacilli: See Bacteria
Bacteremia, 616
Bacteria: See also Diphtheria, bacilli; Meningococci; Milk; Pneumococci; Tubercle Bacilli; Viruses; etc.
abortus and melitense group; urease reaction of Brucella abortus, 597
air contamination and air sterilization, *1023
Bacillus pyocyaneus infection simulating cholera and acute dysentery, 867
Bordet-Gengou: See Whooping Cough
bronchisepticus; Bacillus para-pertussis; species resembling both Bacillus pertussis and Bacillus bronchisepticus but identical with neither, 610

Bacteria—Continued
Coli: See also Urinary Tract, infections
coli, cutaneous sensitivity and its significance, 194
coli; treatment of Bacillus coli infection of urinary tract with mandelic acid, 1341
Corynebacteria: See under Diphtheria
crassus; septicemia due to Diplococcus crassus, simulating typhoid, 412
Culture Mediums: See under names of bacteria
Diphtheria: See under Diphtheria
Dysentery: See Dysentery
Fusiformis: See Angina, Vincent's
Gas: See Gangrene, gas
Hemophilus: See Whooping Cough; etc.
Meningococci: See Meningococci
monocytogenes; identification of Listerella monocytogenes, 1302
monocytogenes; isolation of Listerella monocytogenes from infectious mononucleosis, 1302
Paratyphoid-Enteritidis Group: See Salmonella
Pertussis: See Whooping Cough
Salmonella: See Salmonella
Shiga's: See Dysentery
Sonne's: See Dysentery
Streptococci: See Streptococci
Suipestifer: See under Salmonella
Tuberculosis: See Tubercle Bacilli
Typhoid Group: See Bacteria, coli; Salmonella; Typhoid, bacilli; etc.
Bacteriophage, type-specific, for Corynebacterium diphtheriae, 612
Baker, R. L.: Total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
Total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Bancroft, H.: Active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
Bardet-Laurence-Biedl Syndrome: See Laurence-Biedl Syndrome
Barenberg L. H.: Exanthema subitum (roseola infantum), *983
Barlow's Disease: See Scurvy
Barré-Guillain Syndrome: See Nerves, roots
Basophils: See Pituitary Body
Bates, M. F.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Bauhin's Valve: See Ileocecal Valve
Bayer 205: See Lupus erythematosus
Beets, passage of pigment of red beet into urine of child, 639
Behavior, habit formation, 444
problems of adolescents, *168
Benda, C. E.: Function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
Benjamin, B.: Localized bullous emphysema associated with pneumonia in children, 1139
Benzedrine, Therapy: See under Urination, incontinence
Berenberg, S. R.: Hourglass tumors of spine in children; review of literature and report of case, *131
Beriberi and other symptoms of nutritional disturbance in infant, 182
Besnier-Boeck's Disease: See Sarcoid
Beverly, B. I.: Habit formation, 444
Biedl-Laurence Syndrome: See Laurence-Biedl Syndrome
Biemond Syndrome: See Laurence-Biedl Syndrome
Bile, clinical estimation of bilirubin and biliverdin in bile and in certain fluid mediums, 177
Bile Ducts: See also Gallbladder
anomalies; report of 2 cases with operations and autopsies, *150
Biliary Tract: See Bile Ducts; Gallbladder; Liver
Bilirubin, clinical estimation of bilirubin and biliverdin in bile and in certain fluid mediums, 177
Biliverdin, clinical estimation of bilirubin and biliverdin in bile and in certain fluid mediums, 177
Birth Injury: See Newborn Infants
Premature: See Premature Infants
Rate: See under Vital Statistics
Bismuth and Bismuth Compounds, Therapy: See under Syphilis
Bixby, E. M.: Function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
Blackwater fever following administration of "atebrin," 1318
Bladder: See also Urinary Tract
coexistence of abnormality of vesical neck and bilateral megaureter in child 8 years old; multiple operations; functional cure, 639
congenital valves of posterior urethra, 428
disease of vesical neck in child, 216
inflammation; statistical study of cystitis in children, 1125
rhabdomyosarcoma of urinary bladder, 1339
surgical management of exstrophy of, 1340
Blennorrhea: See Conjunctivitis
Bleyer, A.: Role of advancing maternal age in causing achondroplasia, *994
Blindness with idiocy due to congenital cataracts in 2 brothers, 646
Blood: See also Erythrocytes; Hemoglobin and Hemoglobin Compounds; Hemopoietic System; Leukocytes; under names of diseases; etc.
biochemical studies of blood and spinal fluid in newborn, 1103
Carotene: See Vitamins, A
cholesterol, influence of vitamins C, B_1 and B_2 on, 397
cholesterol; obesity in childhood; basal metabolism and serum cholesterol of obese children, *1001
coagulation and protein spreading, 622
coagulation; normal antithrombin of blood and its relation to heparin, 1325
coagulation; prothrombin in chickens, 870
Diseases: See Anemia; Hemopoietic System; Leukemia; etc.
Globulin: See Blood, proteins
groups; agglutinogens M and N in anthropoid apes, 597
groups; function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
influence of cobalt on iron transportation in blood, 671
Injections: See Blood, transfusion
iodine in leukemia, 1111
iodine, study in childhood, 871
phosphorus and glycerophosphatase of blood of rachitic children; variations observed in course of treatment with ultraviolet radiation, 1097
physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
placental interchange; comparison of total base concentration of fetal and maternal blood at parturition, 858
Platelets: See also Purpura

Blood—Continued
platelets; method of counting thrombocytes in child, 416
pressure, accuracy of clinical determinations in children with values under normal and abnormal conditions, *102
pressure, effect of age on, *113
pressure, high; arterial hypertension in infancy, 873
pressure, low, curability in childhood, 622
pressure, low; orthostatic hypotension; report of 2 cases in which patients were children, 199
proteins; blood coagulation and protein spreading, 622
proteins; generalized edema with hypoproteinemia, 180
proteins; residual nitrogen, total serum protein and albumin-globulin quotient in dyspeptic toxicosis of infants, 1334
proteins; simple method for estimation of total protein content of plasma and serum; estimation of total protein content of human plasma and serum by use of falling drop method, 1111
sedimentation; hemosedimentation of healthy lactating women, 1328
sedimentation; in vitro experiments on effect of added blood serum and blood plasma on sedimentation rate, 1137
sedimentation rates in nontuberculous diseases, 417
sedimentation reaction in otitis media and its complications, 419
sedimentation; rheumatic infections in childhood; effect of surgical operations on blood sedimentation rate, 410
sedimentation time and other blood factors in newborn infant, 185
sedimentation; total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
sedimentation; total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Sugar: See also Carbohydrates; Dextrose; Diabetes Mellitus; Insulin; etc.
sugar; coma due to emotional hypoglycemia in diabetic child, 642
sugar; congenital hypoglycemia, 1304
sugar; epilepsy and alimentary hypoglycemia, 635
sugar; experimental study of cerebral changes in prolonged hypoglycemia, 918
sugar; hematoneurologic syndrome with disturbance of dextrose metabolism from thymicopancreatic dysfunction, 642
sugar; hypoglycemia in 2 year old child with subtotal removal of pancreas, 1367
sugar; influence of lecithin and of lutein on hypoglycemic action of insulin, 642
sugar; observations on blood sugar level before, during and after hunger periods in humans, 627
sugar; pseudohermaphroditism, adiposity, polyuria and hyperglycemia; infundibulotuberian syndrome, 630
sugar; vitamin B_1 and glycemia, 1307
transfusion; accidental transmission of malaria through injection of whole blood, 192
transfusion and rickets, 603
transfusion; capillary bronchitis; its treatment by venoclysis and blood transfusion, 878
transfusion in bronchopneumonia, 1331
transfusion in infants, 621
transfusion in pediatric orthopedics, 1134
transfusion in pediatric therapy, 415
Blood—Continued
transfusion; malaria in infant 7 months old following injections of paternal blood, 193
transfusion; pneumococcic empyema in child and its treatment by ethylhydrocupreine hydrochloride, pneumothorax and blood transfusion, 1331
transfusion; reaction of edematous patient to transfusion with serum or with acacia, 915
transfusion; tanning from ultraviolet irradiation brought out by blood transfusion, *1047
Vessels: See Arteries; Capillaries; Cardiovascular System; Veins; etc.
Vitamins in: See Vitamins
volume; studies in physiology of artificial fever; changes in blood volume and water balance, 1348
volume; studies on volume of circulating blood in children with heart disease, 872
Bloxsom, A.: Pneumonia complicated by acute hemorrhage and ulcerative gastroenteritis in child, *1265
Body Fluids: See Fluids
measurements of Texas school children, 400
special method of measuring physical development ("Minaha" triangle), 605
weight; effect of vitamin B_1 and vitamin B_2 complex on loss of weight produced in rats by experimental hyperthyroidism, 1306
weight; scarlet fever heart, 1316
Boeck's Sarcoid: See Sarcoid
Böttner Test: See Diuresis and Diuretics
Bones: See also under names of bones
atrophy; gargoylism, 606
atrophy; generalized osseous dystrophy, 644
basal metabolism of tuberculous children; tuberculosis of bone, *778
benign bone lesions showing spindle cell invasion of bone, 1128
chemical composition of transverse metaphysial bands produced in growing bones by phosphorus ingestion, 1343
Deformities: See Abnormalities and Deformities; Poliomyelitis; Rickets; etc.
Diseases: See also Osteitis; Osteochondritis; Osteomyelitis; etc.
diseases; review of differential diagnosis of osteopathies due to hormonal causes, avitaminosis and kidney disease, 1345
Dystrophy: See Bones, atrophy; Osteitis fibrosa
end results in patients with severe tuberculosis of bone and with large tuberculous sequestrae, 1323
familial cleidocranial dysostosis (cleidal dysostosis); preosseous and dentinal dystrophy, *786
fragility; brittleness of bones in syphilitic nursling, 414
fragility; experimental scurvy of guinea pig, 603
fragility; syndrome of blue sclerotics (van der Hoeve), 894
growth and development of newborn infants; factors associated with ossification of center for cuboid bone, 678
growth; craniofacial dysostosis (Crouzon's disease), 857
growth; defect of parallelism between osseous growth and dental development considered as factor in etiology of dentofacial malformations, 223
growth; effect of dietary supplement on ossification of bones of wrist in institutional children; effect of cod liver oil supplement, *295
growth; experimental scurvy of guinea pig, 603
growth; sulfur metabolism of early infancy, *37
heterotopic bone formation, 891

Bones—Continued
marrow; acute leukemia (hemocytoblastic), 1327
marrow; diagnosis of kala-azar by search for parasite in bone marrow (puncture of metaphysial region of tibia), 411
marrow; diagnosis of kala-azar by sternal puncture, 1319
marrow; sternal puncture technic and its clinical value with reference to its usefulness in diagnosis of kala-azar, 1108
pathology; aleukemic myelosis with skeletal changes in 7 year old boy, 1112
rate of appearance of ossification centers from birth to age of 5 years, *949
tuberculosis; osteitis tuberculosa multiplex cystoides and sarcoid lesions, 643
tumors; pathology of bone tumors of interest to radiologists, 434
type of osseous dystrophy affecting hands and feet, 218
Bonner, P.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Book Reviews:
Appendicitis: Statistical Study; M. Young and W. T. Russell, 922
Biographies of Child Development; A. Gesell, B. M. Castner, H. Thompson and C. S. Amatruda, 923
Chemistry of Natural Immunity; W. F. Koch, 228
Clinical and Experimental Use of Sulfanilamide, Sulfapyridine and Allied Compounds; P. H. Long and E. A. Bliss, 448
Economical Administration of Health Insurance Benefits: I. Principle of Economy in Administration of Health Benefits; W. Pryll, 683
Einführung in die Kinderheilkunde; E. Glanzmann, 229
Encefalitis postneumonica en el niño; J. Bonaba and C. M. Barberousse, 448
Ensayo de investigacion de la mortalidad infantil en san salvador y algunas sugerencias para combatirla; A. G. Funes and R. Castro, 449
Epidemic Encephalitis: Etiology, Epidemiology, Treatment, 1373
Fragilité osseuse congénitale; H. Fulconis, 923
Infant and Child in Health and Disease: Reference to Nursing Care; J. Zahorsky and E. Noyes, 1149
Intracranial Tumors of Infancy and Childhood; P. Bailey, D. N. Buchanan and P. C. Bucy, 922
Maladies des nourrissons et des enfants; G. Blechmann, 229
Mechanism of Thought, Imagery and Hallucination; J. Rosett, 228
Medical Leaves, 1939: Symposium on Jewish Medical Problems; edited by A. Levinson, 229
Newer Knowledge of Nutrition; E. V. McCollum, E. Orent-Keiles and H. G. Day, 448
Nursing Mental Diseases; H. Bailey, 1149
Patient as Person; G. C. Robinson, 683
Pflege des gesunden und kranken Kindes; W. Catel, 1373
Physiology and Pharmacology of Pituitary Body, Vol. II; H. B. Van Dyke, 447
Pitannya akusherstva i ginekologii; edited by A. M. Gasparyan and P. X. Chajinksy, 449
Pulmonary Tuberculosis in Adults and Children; J. A. Miller and A. Wallgren, 229
Book Reviews—Continued
Raquitismo; J. P. Garrahan, 449
Scarlet Fever; G. F. Dick and G. H. Dick, 447
Seelischer Gesundheitsschutz; H. Meng, 1149
Services des enfants assistés du département de la Seine, 1936; L. Mourier, 446
Services des enfants assistés et de la protection des enfants du premier age du département de la Seine (Premiére partie, statistique; deuxième partie, rapports annuels); L. Mourier, 446
Services des enfants assistés et de la protection des enfants du premier age du département de la Seine (troisième partie, compte financier); L. Mourier, 446
Sex and Internal Secretions: Survey of Recent Research; edited by E. Allen, C. H. Danford and E. A. Doisy, 447
Studien über Vererbung und Entstehung geistiger Störungen. VI. Zur Klinik, Vererbung, Entstehung und Rassenhygiene der angeborenen cerebralen Kinderlähmung (Littleschen Krankheit). Zwillingsbiologische Untersuchungen bei angeborener spastischer Hemi-, Para- und Diplegie (neurologische Zwillingsstudien III. Mitteilung); K. Thums, edited by E. Rüdin, 446
Syndrome hémorragique du nouveau-né; H. Jahier, 922
Syphilis and Its Accomplices in Mischief: Society, State and Physician; G. M. Katsainos, 448
Throat Distemper of 1735-1740; E. Caulfield, 449
Bouillaud's Disease: See Rheumatic Fever
Bourneville-Pringle's Disease: See Sclerosis, tuberous
Boyd, J. D.: Factors in control of dental caries of children, 920
Boyle, H. H.: Concentrated human blood serum as diuretic in nephrosis; further observations, 914
Bradford, W. L.: Effect of hyperimmune human serum (lyophile) and of sulfapyridine on experimental murine pertussis, *1228
Effect of hyperimmune human serum (lyophile) on humoral antibody titer in pertussis, *1234
Brain: See also Cerebellum; Hypothalamus; Lenticular Nucleus; Meninges; Nervous System; Pons Varolii; etc.
abscess observed at ear clinic in Agram, 885
abscess; sulfanilamide in treatment of brain abscess and prevention of meningitis, 899
arachnoiditis of optic chiasm cured by encephalographic examination, 1336
cerebral defect in children born by cesarean section, 1337
cerebral sequelae, after neonatal jaundice, ascribed to kernicterus, 608
congenital morphologic abnormalities of brain in case of abortive tuberous sclerosis; functional implications and bearing on pathogenesis of so-called genuine epilepsy, 1119
cortical form of Heine-Medin's disease, 1315
depth and nerve-cell content of supragranular cortex in normal and mentally defective persons, 426
Diseases: See also Encephalitis
diseases; intracranial complications of ear disease, 624
diseases; leukodystrophia cerebri hereditaria progressiva (Merzbacher-Pelizaeus' disease); 4 cases, 636
embryonic and postembryonic development of cortex, 596
Encephalography: See Brain, roentgenography

Brain—Continued
experimental study of cerebral changes in prolonged hypoglycemia, 918
hemorrhage of brain and retina in scurvy, 600
hemorrhage; precaution in handling of newborn, 1312
hemorrhage; serous apoplexy in children in course of treatment with arsenobenzenes, 631
hemorrhage; serous apoplexy in children; 2 cases, 631
mass and surface development of fetal brain, 885
panel discussion on cyanosis of newborn; cyanosis following intracranial injuries, 1309
roentgenography; cerebral pneumography in childhood, 211
tumors, 885
tumors, and encephalitis, 212
tumors; cerebral tuberculoma, 212
tumors, genetic studies, 887
tumors in young persons, 886
tumors; laterality of signs and symptoms in series of pathologically verified tumors, 211
tumors; ocular signs of intracranial disease in children and juveniles, 1118
urea content of newborn, 1311
Breast Feeding: See Infant Feeding; Milk
hypertrophy; gynecomastia, 251
Milk: See Infant Feeding; Lactation; Milk
Bromer, R. S.: Diagnosis of congenital cardiac defects in infancy; study of 32 cases with necropsies, *8
Bronchi, asthma in children treated with autogenous (bronchoscopic) vaccine, *768
Diseases: See Bronchiectasis; Bronchopneumonia
Bronchiectasis: See also Lungs, cysts
bilateral lobectomy for, 420
congenital abnormalities of lung and acquired bronchiectasis, 878
fatal disease, 1116
lung diseases; pulmonary tuberculosis, mild bronchiectasis, staphylococcal empyema and diaphragmatic hernia into thorax, 420
total pneumonectomy; 2 cases, 626
treatment by intratracheal instillation of kamochin, 207
Bronchitis, Capillary: See Bronchopneumonia
Bronchopneumonia: See also Pneumonia
advances in treatment of infants, 1330
bronchopulmonary staphylococcic infection of newborn; serotherapy, then vaccine therapy; cure, 608
capillary bronchitis; its treatment by venoclysis and blood transfusion, 878
capillary bronchitis of diphtheritic origin, 408
causative organisms in infants in Egypt, 1115
transfusion in, 1331
Bronchoscopy: See Bronchi
Brown, E. W.: Incidence of dysentery in gastroenteritis, 665
Brownlee, I. E.: Total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
Total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Brucella Abortus; Brucella Melitensis: See Bacteria, abortus and melitense group
Bruch, H.: Fröhlich syndrome; report of original case, *1282
Obesity in childhood; basal metabolism and serum cholesterol of obese children, *1001
Obesity in childhood; physical growth and development of obese children, *457
Buddingh, J.: Herpetic stomatitis; enanthem, 907
Bullowa, J. G. M.: Diphtheritic or staphylococcic croup and simultaneous pneumococcic pneumonia, *817
Burns, cancerous scars of, 1134
Butler, A. M.: Determination of ascorbic acid, 667

Cachexia and functional diarrhea; treatment with liver extract, 179
Calciferol: See Vitamins, D
Calcification: See also under Bones, growth; Teeth
arterial calcification and subcutaneous calcinosis; case shown in January 1936, 896
dermatomyositis complicated by calcinosis, 649
Calcinosis: See Calcification
Calcium and Calcium Compounds, comparison of effects of A. T. 10 (dihydrotachysterol) and vitamin D on calcium and phosphorus metabolism in hypoparathyroidism, 600
content of bodies of rats as influenced by that of food; further studies, 1303
continuous calcium and nitrogen balances before and after menarche, 672
Deposits: See Calcification
effect of apples and cranberries on calcium retention, 1093
effect of vitamin D on calcium and phosphorus metabolism; studies on 4 patients, 600
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on calcium retention, *1186
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on nitrogen retention, *965
Gluconate: See Dysentery; Trichinosis
long-time study of nitrogen, calcium and phosphorus metabolism on low protein diet, 1093
long-time study of nitrogen, calcium and phosphorus metabolism on medium protein diet, 1093
requirement of 5 preschool girls, 1094
requirements of infants and children, 1094
Calmette-Guérin (B C G) Vaccine: See under Tuberculosis
Camps, summer, for children, 1309
Cancer: See also Sarcoma; Tumors; and under names of organs and regions
cancerous scars of burns, 1134
Canning and Canned Food, vitamin C content of canned vegetables, 1307
Capillaries, lack of correlation of capillary fragility with vitamin C content of blood, spinal fluid and urine, 198
permeability; verification of tests (Goethlin test) of hypovitaminosis C in children of school age, 1306
Carbohydrates: See also Dextrose; Lactose; etc.
assimilation of protein by young children with nephrotic syndrome; effect of dietary fat and carbohydrate on nitrogen balance, *935
comparative values of various carbohydrates used in infant feeding, 178
Metabolism: See also Blood, sugar; Dextrose; Diabetes Mellitus; etc.
metabolism, experimental studies in intestines, 181
new carbohydrate for prevention of nutritional anemia in infants; preliminary report, *45
pituitary therapy and carbohydrate tolerance, 1369
utilization in human undernutrition, 1094
Carbon Compounds, experimental study of treatment of pneumococcal toxemia with special carbon preparation, 865

Carbon Dioxide: See also Metabolism; Respiration
relative value of pure oxygen and of carbon dioxide mixtures in experimental resuscitation, 858
Carcinoma: See Cancer; and under names of organs and regions
Cardiopathy: See Heart, abnormalities
Cardiovascular System: See also Arteries; Heart; etc.
physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
Carey, B. W.: Incidence of types and therapeutic results in cases of pneumonia of infancy and childhood in Detroit, 658
Caries, Dental: See Teeth
Carotene: See also Vitamins, A
biologic value of carotene in various fats, 1096
Carpus: See Wrist
Carriers: See Diphtheria, carriers; Typhoid, carriers; etc.
Carter, R. F.: Anomalies of bile ducts; report of 2 cases with operations and autopsies, *150
Castration, effect of prepuberal castration of female albino rat on thymus and lymph glands, 1342
effect of thyroidectomy and of adrenalectomy on thymus of castrated white rat, 1342
effect on weight of thymus in adult albino rat, 1343
Catalepsy, cataleptic symptoms in childhood, 1337
Cataract, congenital, 220
congenital familial cataract with cholesterin deposits, 1130
idiocy with blindness due to congenital cataracts in 2 brothers, 646
Catatonia: See Catalepsy; Dementia Praecox
Cecum, correlation of cecum and liver in clinical conditions in children, 629
Celiac Disease, celiac rickets, 209
nutritional problems of childhood, 597
Cerase: See Enzymes
Cerebellum, effect of roentgen irradiation in sarcoma of, 1336
medulloblastoma associated with adiposogenital syndrome, 884
Cerebrospinal Fever: See Meningitis
Cerebrospinal Fluid: See also Spinal Puncture
biochemical studies of blood and spinal fluid in newborn, 1103
colloidal carbon reaction of, 1110
differentiation between meningitis and encephalitis by color reaction of, 1337
in normal newborn infants and infants with meningeal hemorrhage, 608
neurolytic ferments in spinal fluid of healthy and sick children, 1337
of syphilitic children, 414
polyradiculoneuritis with dissociation in protein content and cellular content of, 883
vitamin C content of, 1336
vitamin C in spinal fluid during hunger, 603
Cerebrum: See Brain
Cesarean Section; cerebral defect in children born by cesarean section, 1337
Cevitamic Acid: See Vitamins, C
Chattás, A.: Tuberculosis in children less than 6 years of age, *92
Chauffard-Still Disease: See Arthritis deformans
Chest: See Thorax
Chickenpox, embolus of femoral artery following chickenpox, 1314
encephalitis with varicella; 3 cases, 862
hemorrhagic varicella in infant, 613
treatment of severe varicella with injection of convalescent blood, 862
varicella encephalitis, 1106
Child Welfare: See also Infant Welfare
fifteen years of work of Polish committee on child care and scientific publications, 606
maternal and child health services under Social Security Act, 1100
maternal and child health under National Health Program, 903
needed analyses of child welfare activities, 403
psychology, baby health and child welfare, 213
recommendations issued by fourteenth Congress of Hygiene, 399
Childe, A. E.: Localized bullous emphysema associated with pneumonia in children, 1139
Children: See also Infants; Newborn Infants; Pediatrics; Schools; etc.
bringing up and teaching of children in institutions for sick, 427
disabling diseases of childhood; their characteristics and medical care as observed in 500,000 children in 83 cities canvassed in National Health Survey of 1935-1936, *1157
education and training; problems in child guidance from point of view of pediatrician, 427
education and training; reward as aid for child in training, 427
Growth: See Growth
Nervous and Mental Disabilities: See Nervous and Mental Disabilities
physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
play-interview; method of studying children's attitudes, *1199
training of preschool child, 1122
China and Chinese, anterior poliomyelitis in China, 408
phlyctenular disease in Shanghai, 895
studies on Hunan local food products; chemical composition and vitamin C content of Hunan citrus fruits, 604
studies on Hunan local food products; vitamin C content of "vegetable water," 397
survey of intestinal parasites in Chinese hospital patients in Shanghai, 413
Chloroma, congenital leukemia with "chloroma," *332
myelosarcoma simulating chloroma, 1112
Cholecystitis: See Gallbladder
Cholelithiasis: See Gallbladder
Cholera, Bacillus pyocyaneus infection simulating cholera and acute dysentery, 867
Cholesterol: See also Lipoids
cholesterinosis bulbi, 437
congenital familial cataract with cholesterin deposits, 1130
function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
in Blood: See Blood, cholesterol
Chondrodystrophia Fetalis: See Achondroplasia
Chondrodystrophy: See Achondroplasia
Chondro-Osteodystrophy: See Bones, atrophy
Chorea and athetosis in childhood, 1119
effect of sulfanilamide on rheumatic fever and chorea, 615
etiology; its relation to rheumatic fever and heart disease, 424
incidence of rheumatic infections in children of Oregon, 410
minor, etiologic factors in, 426
minor, further studies on problem; clinical, etiologic and pathologic aspects, 633
minor, treatment of, 1121
treatment with magnesium sulfate, 884
use of fever therapy in children, 651
Choriomeningitis: See Meningitis
Christian-Schüller Syndrome: See Schüller-Christian Syndrome

Christie, A. U.: Growth and development of newborn infants; factors associated with ossification of center for cuboid bone, 678
Cicatrix, cancerous scars of burns, 1134
Circulatory System: See Cardiovascular System
Circumcision, local anesthesia in children, 1134
Clarke, H. T.: Late rickets with glycosuria and organic acid acidosis (Fanconi type), 673
Cleft Palate: See Palate
Clifton, W. M.: Hyperparathyroidism in children; report of 2 cases, *540
Climate, effect on convalescent children, 1136
Clinocephaly: See Cranium, abnormalities
Cobalt, Therapy: See Anemia
Coccidioides, acute coccidioidomycosis with erythema nodosum in children, 902
Cod Liver Oil, effect of dietary supplement on ossification of bones of wrist in institutional children; effect of cod liver oil supplement, *295
Cohen, P.: Anemia of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
Cohen, W.: Total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *81
Total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Colds: See Respiratory Tract, diseases
Colic, Lead: See Lead, poisoning
Umbilical: See Umbilicus
Collins, H. L.: Anomalies of bile ducts; report of 2 cases with operations and autopsies, *150
Colon: See also Cecum; Gastrointestinal Tract; Intestines; Sigmoid
abnormalities; congenital microcolon, 423
dilatation; congenital megacolon in boy of 10 years; improvement following operation on sympathetic nervous system, 423
dilatation; congenital megacolon in child with mongolism, 629
dilatation; surgical treatment of congenital megacolon, 1116
melanosis coli in boy aged 2½ years, 208
Colostrum, antibacterial properties of, 1303
colostrum bodies, 1095
Coma: See under Diabetes Mellitus
Communicable Diseases: See also Exanthems; Immunity; Measles; Meningitis; Syphilis; etc.
case for individual isolation; its use in infectious diseases and in care of infants, 1314
cutaneous manifestations of acute exanthematous diseases, 188
disabling diseases of childhood; their characteristics and medical care as observed in 500,000 children in 83 cities canvassed in National Health Survey of 1935-1936, *1157
infectious diseases in 1938, 862
local and peripheral leukocyte counts in certain infectious diseases, 1315
prevalence in United States, 861, 1314
prophylactic and therapeutic value of placental extract in regard to measles, scarlatina and mumps, 190
recent outbreaks of, 861
sequelae of postinfectional encephalitis with reference to mental involvement, 1352
some limitations in preventive medicine, 1347
summary of current prevalence, 405
therapy with convalescent human serum, 1105
Complement, ascorbic acid and complement function, 601
Complement—Continued
complementing activity and ascorbic acid content of guinea pig serums following ether anesthesia, 601
effect of ascorbic acid on constitution of, 601
in newborn children, in infants and in fetuses, 860
Conjunctivitis: See also Keratoconjunctivitis
inclusion blennorrhea, 1102
phlyctenular disease in Shanghai, 895
Conn, J. H.: Factors influencing development of sexual attitudes and sexual awareness in children, *738
Play-interview; method of studying children's attitudes, *1199
Constitution; constitutional factors in etiology of prematurity, 1101
Contagious Diseases: See Communicable Diseases
Convalescence, effect of climate on convalescent children, 1136
Convulsions: See also Eclampsia; Epilepsy; etc.
experimental studies concerning pathology of insulin convulsions, 918
Cooley, T. B.: Incidence of types and therapeutic results in cases of pneumonia of infancy and childhood in Detroit, 658
Cooley Syndrome: See Pseudoleukemia, infantile
Cooper, M. L.: Incidence of dysentery in gastroenteritis, 665
Copper content of human milk, 180
content of milk in anemic infants, 181
Corn, further report on corn rust poisoning in childhood, 1136
Corn Syrup, comparative values of various carbohydrates used in infant feeding, 178
Corynebacteria: See under Diphtheria
Coughing and straining, effect on blood pressure, *115
Cowie, D. M.: Distribution and content of iodide in iodized salt obtained on open market throughout Michigan, 1370
Thymus and thymicolymphatic constitution: introductory remarks, 1357
Cranberries, effect of apples and cranberries on calcium retention, 1093
Craniotabes, 1344
Cranium: See also Temporal Bone; etc.
Abnormalities: See also Acrocephaly
abnormalities; hypoplasia of cerebral motor nuclei or nerves, combined with clinocephaly, 1120
cranio-carpo-tarsal dystrophy of ? undescribed type; 2 cases, 217
familial cleidocranial dysostosis (cleidal dysostosis); preosseous and dentinal dystrophy, *786
ocular signs of intracranial disease in children and juveniles, 1118
pathology; histologic examination of hypothalamus in ordinary obesity, in Morgagni's and Günther's syndromes and in morbus Cushing, 1120
Creatine and Creatinine in Urine: See under Urine
new creatinine standard for basal metabolism and its clinical application, *506
Cretinism: See also Myxedema
sporadic, two cases of complete recovery, 910
Cripples: See also Abnormalities and Deformities
school life and after for handicapped child, 1099
Cross, A. R.: Congenital bilateral radio-ulnar synostosis, *1259
Croup, diphtheritic or staphylococcic croup and simultaneous pneumococcic pneumonia, *817
Crouzon's Disease: See Bones, growth
Crump, J.: Asthma in children treated with autogenous (bronchoscopic) vaccine, *768

Cryptococci Meningitidis: See Torula
Cryptorchidism: See Testicles, undescended
Crystalline Lens: See Lens, Crystalline
Cubitus: See Forearm
Cullen, G. E.: Incidence of dysentery in gastroenteritis, 665
Culture Mediums: See under Bacteria; etc.
Cuneiform Bone: See Tarsus
Curnen, E. C., Jr.: Serum treatment of pneumonia due to pneumococcus type XIV, 656
Curphey, T. J.: Unusual fatal perforating wound of chest, *1278
Curtis, G. M.: Acute suppurative thyroiditis during childhood, *837
Cushing Syndrome: See Pituitary Body, diseases
Cushman, M.: Determination of ascorbic acid, 667
Cyanosis, circulatory causes; panel discussion, 1310
congenital cardiopathy without murmurs, 1327
cyanosis following intracranial injuries; panel discussion, 1309
in newborn, 403
in newborn; panel discussion, 1309
multiple pregnancies in woman with congenital cyanosis due to heart disease, 1326
obstetric aspects; panel discussion, 1311
pathologic aspects; panel discussion, 1310
pulmonary causes; panel discussion, 1310
Cystine and Cysteine, syndrome consisting of affections of kidney, stunted growth, rickets and disturbed cystine metabolism, 215
Therapy: See Arthritis deformans
Cystitis: See Bladder, inflammation
Cysts: See under names of organs and regions, as Intestines; Lungs; Vagina; etc.
Echinococcus: See Echinococcosis
Epidermoid: See Temporal Bone
Hydatid: See Echinococcosis

Dark Adaptation: See Eyes, accommodation and refraction
Davison, C.: Erythroblastic (Cooley's) anemia and neurologic complications (status dysmyelinatus), *362
Day, R.: Effect of sleep on insensible perspiration in infants and children, *82
Deaf-Mutism, deaf child, 625
Deafness: See also Otitis Media
syndrome of blue sclerotics (van der Hoeve), 894
unilateral, following antidiphtheritic vaccination, 862
Death, Rate: See Infant Mortality; Vital Statistics
sudden, of boy of 12 years, 622
Defectives: See Cripples; Feeblemindedness; Nervous and Mental Disabilities
Deficiency Diseases: See also Diet and Dietetics; Nutrition; Vitamins; etc.; and under names of various diseases, as Beriberi; Scurvy; etc.
newer aspects of nervous disorders in avitaminosis, 852
Deformities: See Abnormalities and Deformities; and under names of organs and regions
Dehydration, acute, without toxic symptoms, 598
dehydrating therapy in cases of infantile paralysis, 1317
Delinquency, girls on parole and after, 442
Dementia: See also Dementia Paralytica; Dementia Praecox; etc.
outcome of convulsions in early childhood and effect of roentgen rays, 425
Dementia Paralytica, general paralysis in mother and daughter, 619
Dementia Praecox, mental disturbances in children resembling schizophrenia, 638
schizophrenia beginning at about 3 years of age, 214
Dementia Praecox—Continued
schizophrenia-like psychoses in young children, 637
schizophreniform psychoses in childhood, 637
Demography: See Infant Mortality; Vital Statistics
Dentition: See Teeth
Dermatitis: See also Eczema; etc.
contact, 648
exfoliativa; Ritter's disease, 186
from contact with garlic, 439
Medicamentosa: See under names of drugs
Dermatomyositis complicated by calcinosis, 649
Dextrimaltose, comparative values of various carbohydrates used in infant feeding, 178
Dextrocardia: See Heart, displacement
Dextrose: See also Blood, sugar; Carbohydrates
comparative values of various carbohydrates used in infant feeding, 178
late rickets with glycosuria and organic acid acidosis (Fanconi type), 673
Diabetes Mellitus: See also Blood, sugar; Insulin
coma due to emotional hypoglycemia in diabetic child, 642
comparative studies on use of standardized protamine zinc and crystalline insulin, 919
dietetic and insulin management of juvenile diabetic, 431
free diet method of dealing with diabetes mellitus in childhood, 643
juvenile, 890
lipodystrophy and lipomatosis from repeated injections of insulin, 1341
local lipomatosis caused by insulin injections, 1343
pituitary therapy and carbohydrate tolerance, 1369
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Diaphragm, Hernia: See Hernia, diaphragmatic
traumatic rupture in child, 223
Diarrhea: See also Dysentery
epidemic, of newborn, 662
functional, with cachexia; treatment with liver extract, 179
predisposition to, 210
Salmonella panama, 421
some aspects in newborn infant, 186
treatment in children, 421
winter diarrhea in infants, 879
Diathermy: See under names of diseases, organs and regions
Diathesis, Exudative: See Eczema
Hemorrhagic: See also Hemophilia; Purpura; Scurvy; etc.
hemorrhagic, vitamin C therapy, 1327
Dick Test: See under Scarlet Fever
Dickson, E. C.: Acute coccidioidomycosis with erythema nodosum in children, 902
Diet and Dietetics: See also Food; Infant Feeding; Nutrition; Vitamins; and under names of various diseases and foods
diet and its relation to dental disease, 1134
diet in health and disease; general principles of diet in childhood, 395
diet in health and disease; special diets in childhood, 1303
effect of dietary supplement on ossification of bones of wrist in institutional children; effect of cod liver oil supplement, *295
relation of diet to oral health, 222
Digestive Tract: See also Gastrointestinal Tract; Intestines; Stomach; etc.
digestibility of raw potato starch in man, 849
epidemic of alimentary intoxication due to organism of paratyphoid B group, 1333

Digitalis Therapy: See under names of various diseases, as Diphtheria; Heart, disease
Dihydrotachysterol, Therapy: See Spasmophilia
Dilantin, Therapy: See Epilepsy
Diphtheria and bacterial types of Corynebacterium diphtheriae, 612
capillary bronchitis of diphtheritic origin, 408
carriers, vaccine treatment, 191
comparative studies on purification of tetanus and diphtheria toxins, 847
demonstration of free diphtheria toxin in patients' blood, 864
diphtheritic involvement of lips with absence of signs in nose and throat, 187
diphtheritic or staphylococcic croup and simultaneous pneumococcic pneumonia, *817
effect of vitamin C on Schick reaction, 603
experimental, metabolism in dog, 408
immunization; optimum spacing of doses of alum precipitated toxoid, 189
in Lübeck from 1910 to 1937, 1315
in Lübeck from 1937 to 1938, 1316
in Nuremberg in recent years, 1315
influence on measurements of heart and alterations of heart muscle tissue, 408
investigation concerning QT interval of electrocardiogram in diphtheria of children, 416
laryngeal; laboratory diagnosis of malignant diphtheritic laryngitis, 1107
myocardial changes in, 863
nasal, and tonsillectomy, 864
nasal spraying with zinc sulfate solution in prophylaxis of poliomyelitis; diphtheria immunization as partial preventive, 190
Paralysis Following: See under Paralysis
prevention, *512, 861
relation between sunspots and diphtheria, 1316
Schick testing and carrier rates after diphtheria immunization, 189
serum disease, 898
studies in immunity; spontaneous changes in Dick and Schick reactions of children, *734
study of types of Corynebacterium diphtheriae, 860
suppurative streptococcic lymphadenitis following diphtheria in brothers, 864
things of practical importance in, 1107
treatment of diphtheria and scarlet fever by active immunization, 406
type-specific bacteriophages for Corynebacterium diphtheriae, 612
unilateral deafness following antidiphtheritic vaccination, 862
value of electrocardiography in, 1315
value of 1 injection of alum precipitated toxoid in controlling diphtheria, 187
vitamin C nutrition and susceptibility to diphtheria as possible factors in epidemiology of poliomyelitis, 1314
when shall digitalis be given in diphtheria? 1315
Diplococcus Crassus: See Bacteria, crassus
Directory of Medical Specialists, 1090
Directory of pediatric societies, 230, 450, 684, 924, 1150, 1374
Disease: See also Constitution; Medicine; etc.
in twins, 184
Disinfection and Disinfectants: See also Sterilization
control of impetigo, *931
Dislocations: See under names of joints, as Hip Joint; etc.
Diuresis and Diuretics: See also Kidneys; Water; etc.
Böttner and Kauffmann method of determining heart function, 1112
concentrated human blood serum as diuretic in nephrosis; further observations, 914
Diverticulum, Meckel's: See under Intestines
Dodd, K.: Herpetic stomatitis; enanthem, 907
Dolichostenomelia: See Arachnodactylia; Extremities, abnormalities
Doull, J. A.: Active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
Doyle, M. E.: Air contamination and air sterilization, *1023
Drinker Respirator: See Respiration, artificial
Drugs: See under names of individual drugs and under names of diseases
Ductus Arteriosus: See Heart, abnormalities
Dunham, E. C.: Growth and development of newborn infants; factors associated with ossification of center for cuboid bone, 678
Duodenum, stenosis in newborn infant, 1118
Ulcer: See Peptic Ulcer
Dwarfism: See also Achondroplasia
changes of temporal bone in rickets and in "renal rickets," 1098
pituitary dwarfism treated with antuitrin-G, 430
syndrome consisting of affections of kidney, stunted growth, rickets and disturbed cystine metabolism, 215
Dysentery: See also Diarrhea
Amebic: See Amebiasis
bacillary, calcium gluconate and kaolin in treatment, 191
bacillary, in young infants, 1321
bacillary, outbreak in children's camp, 865
bacilli; para-Shiga dysentery bacillus, 1108
Bacillus pyocyaneus infection simulating cholera and acute dysentery, 867
in infancy, 195
incidence in gastroenteritis, 665
infection of urinary tract with Bacillus dysenteriae, *830
otologic relations to dysentery in infancy, 1319
Dysostosis: See Bones, growth
Dyspepsia: See also Celiac Disease; Gastrointestinal Tract, diseases
agar milk in dyspepsia and enterocolitis, 1334
residual nitrogen, total serum protein and albumin-globulin quotient in dyspeptic toxicosis of infants, 1334
Dysphonia: See Voice
Dyspnea, 877. See also Asthma
Dystrophy: See also Bones, atrophy; etc.
Adiposogenital: See Laurence-Biedl Syndrome; Pituitary Body
familial cleidocranial dysostosis (cleidal dysostosis); preosseous and dentinal dystrophy, *786
Muscular: See also Myotonia
muscular; atonic-sclerotic muscular dystrophy (type Ulrich), 1345
muscular atrophies and dystrophies in childhood, 434
muscular; hereditary muscular atrophy (Leyden-Moebius type) treated with various aminoacids, 1344
muscular; is there uniform heredity in progressive muscular dystrophy? 892
muscular; muscle dystrophies and atrophies occurring in childhood from orthopedic aspect, 441
muscular, pseudohypertrophic, 433
muscular; significance of creatinuria in progressive muscular dystrophy, 892

Ear: See also Deafness; Mastoiditis; etc.
Diseases: See Meningitis, otitic; Otitis Media
does animal experimentation show similar changes in ear of mother and fetus after ingestion of quinine by mother? 605
use of Mayer position in roentgenographic examination of ear of newborn infant, 185
Ebbs, J. H.: Comparative incidence of infections in breast fed and artificially fed infants, 1146

Echinococcosis: See also under names of various organs
hydatid cyst of spinal canal successfully treated by operation, 425
hydatid cysts of lung, 1331
Eclampsia, eclampsia-like syndrome occurring in pregnant dogs and rabbits following renal artery constriction, 1339
with icterus gravis in child during first year of life, 629
Economic Conditions, nutrition as affected by economic level, 1098
Ecthyma in year old child, 1133
phagedenic, in infant, 439
Ectodermal Defect, congenital ectodermoses in infancy and childhood, 1346
two families showing hereditary ectodermal dystrophies, 896
Ectopia Cordis: See Heart, displacement
Eczema, acute accidents in eczematous infants, 439
chronic, treatment in children, 1145
contact and environmental allergens as cause, 437
dietetic treatment of "eczema" in early infancy, 648
flavine dye (rivanol) in noninfectious eczema of childhood, 1133
immunologic relations between cow's milk and goat's milk, 905
incompatibility of mother's milk obtained during menstruation in infant with exudative diathesis, 1133
infantile, diagnosis of, 1347
infantile, treatment, 895
infantile, treatment from point of view of allergist, 896
rapid death in infancy; role of neurovegetative system, 439
Edema: See also under Brain; Lungs; etc.
anasarca due to nephrosis and intercurrent erysipeloid cutaneous lesions, 640
anemia of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
avitaminotic, of infancy, 440
generalized, with hypoproteinemia, 180
nutritional, in children in Egypt, 395
reaction of edematous patient to transfusion with serum or with acacia, 915
Education and Training: See also under Children; Schools
education in early infancy, 214
Edwards, H. E.: Treatment of chronic eczema in children, 1145
Electrocardiogram: See under Heart
Electrocoagulation: See under specific headings, as Tonsillectomy; etc.
Eliot, M. M.: Maternal and child health under National Health Program, 903
Ellis, D. M.: Total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
Total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Embolism: See also Thrombosis
embolus of femoral artery following chickenpox, 1314
Embryology: See Fetus; Monsters; Teratoma; and under names of special organs and structures
Embryoma, Wilms', 1124
Emerson, L.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Emesis: See Vomiting
Emotions, come due to emotional hypoglycemia in diabetic child, 642
emotional and nervous diseases in pediatrics from clinical and social points of view, 1339
toward development of emotional stability, 1121
Emphysema, erroneous diagnosis of bullous emphysema of child's lung, 1331
interstitial, in asthmatic patient, 1116
localized bullous emphysema associated with pneumonia in children, 1139
Empyema, action of intrapleural injections of hypertonic solutions of sugar in pleuritis exsudativa and in empyema, 421
due to Staphylococcus aureus in infant 2 months old; treatment with staphylococcus anatoxin; cure, 626
lung diseases; pulmonary tuberculosis, mild bronchiectasis, staphylococcal empyema and diaphragmatic hernia into thorax, 420
observations on treatment in children, 877
pneumococcic, in child and its treatment by ethylhydrocupreine hydrochloride, pneumothorax and blood transfusion, 1331
treatment, 207
Encephalitis: See also Encephalomyelitis; Meningoencephalitis
acute, of whooping cough, 1106
and tumor of brain, 212
central lobar leukoencephalosclerosis, symmetrical, primary and progressive, 425
cerebral complications in scarlet fever, 863
diagnosis in infancy, 633
differential diagnosis of pontile tumors and encephalitis, 1336
differentiation between meningitis and encephalitis by color reaction of spinal fluid, 1337
disturbance of gait, probably following scarlatinal encephalitis, 1314
human encephalitis; 8 fatal cases with 4 due to virus of equine encephalomyelitis, 1119
sequelae of postinfectional encephalitis with reference to mental involvement, 1352
therapy of encephalitis and related neurotropic infections of nervous system, 1120
varicella encephalitis, 1106
with varicella; 3 cases, 862
Encephalitozoon, granulomatous encephalomyelitis due to protozoan (Toxoplasma or Encephalitozoon); identification of case from literature, 1335
Encephalography: See Brain, roentgenography
Encephalomeningitis: See Meningoencephalitis
Encephalomyelitis, granulomatous, due to protozoan (Toxoplasma or Encephalitozoon); identification of case from literature, 1335
human encephalitis; 8 fatal cases with 4 due to virus of equine encephalomyelitis, 1119
Encopresis: See Feces, incontinence
Endocarditis, meningococcic, and myocarditis, *823
Rheumatic: See under Rheumatic Fever
treatment of subacute bacterial endocarditis with M. & B. 693, 200
tuberculous, of pulmonary valve, 871
Endocrine Glands: See also Hormones; Metabolism; and under names of glands
juvenile femoral osteochondritis with endocrine difficulties, 1129
review of differential diagnosis of osteopathies due to hormonal causes, avitaminosis and kidney disease, 1345
Endocrine Therapy: See under names of diseases, glands, hormones and organs
Enemata, mercurial poisoning from bichloride of mercury enema, 651
Energy Exchange: See Metabolism
Engelfried, J. J.: Comparison of different methods in use for determination of sulfanilamide in tissues and fluids of body, 1364

Enteritis: See under Intestines
Enterocolitis: See Intestines, diseases
Enuresis: See Urination, incontinence
Enzymes: See also under names of enzymes, as Urease; etc.
moth cerase and tubercle bacilli, 619
neurolytic ferments in spinal fluid of healthy and sick children, 1337
oral administration of hormonal proteins, 888
Eosinophilia in premature infants, 1327
syndrome of Löffler, 878
Eosinophils: See also Leukocytes
eosinophilic bodies in blood of mumps, 861
Ephedrine, Therapy: See under Nose
Epidermis: See Skin
Epidermoids: See Temporal Bone
Epidermolysis bullosa, 649
Epilepsy: See also Convulsions; Sclerosis, tuberous
and alimentary hypoglycemia, 635
and heredity; problem of epileptoid psychopathy, 887
congenital morphologic abnormalities of brain in case of abortive tuberous sclerosis: functional implications and bearing on pathogenesis of so-called genuine epilepsy, 1119
diagnostic differentiation from pyknolepsy and salaam convulsions in children, 884
genetic studies of children of epileptic parents, 634
heredity in, 883
heredity in; study of children of epileptic patients, 633
mentality of dispensary epileptic patients, 213
neurohumoral nature of, 1335
outcome of convulsions in early childhood and effect of roentgen rays, 425
preliminary observations on dilantin therapy of convulsions, 1143
Epilola: See Sclerosis, tuberous
Epinephrine: See Adrenal Preparations
Epiphyses, age order of epiphysial union in Bengalee girls, 644
traumatic separation of upper epiphysis of femur; typical birth injury, 609
Epithelioma: See also Cancer
calcified and mummified epitheliomas of skin in children, 897
Ergosterol, Irradiated: See Viosterol
Erysipelas, prognosis and treatment (with sulfanilamide), 191
treatment in infants with sulfanilamide, 613
Erythema exsudativum multiforme; meningococcic sepsis running course of erythema exsudativum multiforme with subsequent meningitis, 864
nodosum with acute coccidioidomycosis in children, 902
Erythremia: See Polycythemia
Erythroblastosis: See Anemia; Hemopoietic System; Pseudoleukemia, infantile
Erythrocytes: See also Anemia; Blood; Hemoglobin and Hemoglobin Compounds; Polycythemia; etc.
appearance of reticulocytes in scarlet fever, 409
clinical studies of behavior of reticulocyte count, 873
clinical study of reticulocyte count in blood diseases of children, 873
congenital hemolytic anemia with normal fragility of red blood cells, 403
erythrocytometric formula, number of red cells and of reticulocytes in newborn and their variation in number in first days of life, 859
hematocrit determinations in normal and abnormal blood, 415
reticulocytes in measles, 409
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Erythrocytes—Continued
Sedimentation: See Blood, sedimentation
Esophagus, achalasia, 880
congenital atresia of, 1333
esophagitis in infancy, 209
Foreign Bodies: See under Foreign Bodies
pleural manifestations of perforation, *1261
stricture, and lye poisoning, 1117
Estrogens: See also Hormones, sex; Ovary, hormone; Pregnancy, urine in
clinical use of new estrogen, 890
excretion and fate of androgens, 641
treatment of gonorrheal vaginitis with estrogen, 217
treatment of grave condition in premature infant with estrogen, 402
Ether Anesthesia: See under Anesthesia
Ethylhydrocupreine: See Empyema
Exanthem Subitum: See Roseola
Exanthems: See also Communicable Diseases; and under names of exanthematous diseases
eruptive fever (dengue, measles, German measles?) in reality complex toxic drug dermatitis (pentavalent arsenic and sulfanilamide derivative), 650
Excitement, effect on blood pressure, *115
Exercise, physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
Extrasystole: See Arrythmia
Extremities, abnormalities; dislocation of ocular lenses of premature infant; dolichostenomelia? 646
congenital abnormalities of extremities; hands, 1100
Gangrene: See Gangrene
Paralysis: See Poliomyelitis
paroxysmal myopathic crises with hemoglobinuria, 645
Eyes: See also Orbit; Vision; and under names of special structures and diseases of eyes
accommodation and refraction; vitamin A deficiency in disease of thyroid gland; its detection by dark adaptation, 1127
diseases, external, in school children, 645
diseases; ocular complications in rheumatism, 1132
diseases; present status of shock and fever therapy in ophthalmology, 893
melanosis; familial congenital unilateral melanosis, 220
ocular defects as cause of migraine in children, 220
ocular manifestations of congenital syphilis, 219
ocular signs of intracranial disease in children and juveniles, 1118
pathology; cholesterinosis bulbi, 437

Faber, H. K.: Acute coccidioidomycosis with erythema nodosum in children, 902
Face, congenital hypertrophy of right side of body and left side of face, 184
Paralysis: See Paralysis, facial
Failmezger, T. R.: Diagnosis of trichinosis, *129
Fallot's Tetralogy: See Heart, abnormalities
Farr, L. E.: Assimilation of protein by young children with nephrotic syndrome; effect of dietary fat and carbohydrate on nitrogen balance, *935
Assimilation of protein by young children with nephrotic syndrome; effect of nephrotic crises on assimilation of nitrogen, *939
Fat: See also Acid, fatty; Lipoids
assimilation of protein by young children with nephrotic syndrome; effect of dietary fat and carbohydrate on nitrogen balance, *935
biologic value of carotene in various fats, 1096

Fat—Continued
in Milk: See under Milk
lipodystrophy and lipomatosis from repeated injections of insulin, 1341
local lipomatosis caused by insulin injections, 1343
metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
note on vitamin B_1—sparing action of fat, 1096
studies of fat-free diets, 1093
subcutaneous adiponecrosis and lipogranulomatosis in infant, 222
Fatigue, recommendations issued by fourteenth Congress of Hygiene, 399
Favism in childhood, 1349
Feces, changes brought about in qualitative composition of bacterial flora in infants' stools by citric acid milk, 396
citric acid fermentation by bacteria, 396
incontinence in children; 20 cases, 422
involuntary defecation in 9 year old girl with congenital syphilis, 870
Feeblemindedness: See also Idiocy; Laurence-Biedl Syndrome; etc.
does birth of mentally defective children show seasonal fluctuation? 1339
school life and after for handicapped child, 1099
sequelae of postinfectional encephalitis with reference to mental involvement, 1352
syndrome characterized by progressive cerebellar ataxia with feeblemindedness, 632
Feet: See Foot
Feil-Klippel Syndrome: See Spine, abnormalities
Felsen, J.: Infection of urinary tract with Bacillus dysenteriae, *830
Femur, Epiphyses: See Epiphyses
Fenton, M. M.: Juvenile asthma, 1365
Fetus: See also Labor; Monsters; Pregnancy; Umbilical Cord
biometric study of growth in premature infant, 1101
complement in newborn children, in infants and in fetuses, 860
correlation of fetal body length with weights of organs, 1102
death of; Spalding's sign of intrauterine death of fetus, 1101
evidence of fetal respiration in utero, 1308
fetal heart sounds studied by phonocardiograph, 872
fetal peritonitis, 857
flying fetus, 857
human fetal electrocardiogram, 184
mass and surface development of fetal brain, 885
Pathology: See also Achondroplasia; Hydrocephalus; etc.
pathology; genesis of congenital hydrops, 1103
pathology; hydrops foetalis (erythroblastosis), 607
placental interchange; comparison of total base concentration of fetal and maternal blood at parturition, 858
teratoma of neck of fetus with acute hydramnios, 1308
Fever: See also Malaria; Temperature; Typhoid; etc.
artificial, studies in physiology of; changes in blood volume and water balance, 1348
artificial, vitamin C nutrition in, 1305
athetosis following acute specific fevers, 193
Eruptive: See Exanthems
hyperpyrexia following operation for cleft lip or palate, *320
Therapeutic: See under Chorea; Eyes, diseases; etc.
Finger Sucking and thumb sucking in childhood, *1290
Fingers and Toes: See also Foot; Hand
Abnormalities: See also Arachnodactylia abnormalities; craniofacial dysostosis, 857
developmental abbreviation of terminal phalanges, 857
Fistula, aural; retropharyngeal abscess with fistulization into external auditory canal, 874
Flavine Dye: See Eczema
Flood, R. G.: Control of impetigo, *931
Fluids, clinical estimation of bilirubin and biliverdin in bile and in certain fluid mediums, 177
comparison of different methods in use for determination of sulfanilamide in tissues and fluids of body, 1364
effect of thirst and salt deficiency on water depots of organism, 1304
extracellular and intracellular fluid, 852
Folsom, T. G.: Epidemic diarrhea of newborn, 662
Food: See also Diet and Dietetics; Nutrition; Vitamins; and under various foods, as Apples; Milk; Vegetables; etc.
allergy, in childhood, 221
early introduction of solid foods in infant diet, 1144
Poisoning: See also Paratyphoid; Salmonella poisoning; Salmonella panama, 421
Foot: See also Fingers and Toes; Tarsus; etc.
type of osseous dystrophy affecting hands and feet, 218
Forceps: See Labor
Forearm, syndrome of infantilism, congenital webbed neck and cubitus valgus, 856
Foreign Bodies: See also under names of organs and regions
in upper esophagus for 5 months, 879
Fractures: See under Bones, fragility; Pelvis; etc.
Freireich, A. W.: Unusual fatal perforating wound of chest, *1278
Friedman, T. B.: Role of allergy in atelectasis in children, *237
Friedreich's Ataxia: See Ataxia, hereditary
Fröhlich Syndrome: See Laurence-Biedl Syndrome; Pituitary Body
Frontal Sinus: See also Sinuses, Nasal
anatomic consideration of development, 204
Fruit, administration of vitamin C in form of ascorbic acid and of fruit juice, 856
studies on Hunan local food products; chemical composition and vitamin C content of Hunan citrus fruits, 604
Fungi, preparation of mold antigens for diagnostic and therapeutic purposes, 1367
Furcolow, M. L.: Incidence of dysentery in gastroenteritis, 665
Furstenberg, A. C.: Thymus and thymicolymphatic constitution; clinical aspects as seen by otolaryngologist, 1359
Fusospirilliosis: See Angina, Vincent's

Gait: See Locomotion
Galactose Tolerance: See Liver, function
Gallbladder: See also Bile; Bile Ducts
cholecystitis and cholelithiasis in childhood, 421
Gangrene: See also under names of organs and regions
cutaneous familial infantile gangrene, 897
gas, 411
Gargoylism: See Bones, atrophy
Garlic, dermatitis from contact with garlic, 439
Gas Gangrene: See Gangrene
Gastroenteritis: See Gastrointestinal Tract
Gastrointestinal Tract: See also Colon; Digestive Tract; Intestines; Rectum; Stomach; etc.

Gastrointestinal Tract—Continued
comparative incidence of infections in breast fed and artificially fed infants, 1146
detection of virus of poliomyelitis in nose and throat and gastrointestinal tract of humans and monkeys, 610
diseases; value of raw apple treatment, 423
hemorrhage of newborn; 2 cases, 1311
incidence of dysentery in gastroenteritis, 665
nutritional edema in children in Egypt, 395
perforation of newborn infant, *371
pneumonia complicated by acute hemorrhage and ulcerative gastroenteritis in child, *1265
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Gaucher's Disease: See Anemia, splenic
Gedgoud, J. L.: Physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
Gee's Disease: See Celiac Disease
Genitals: See also Genitourinary Tract; Gynecology; Urinary Tract; and under names of genitals, as Testicles; Vagina; etc.
effect of prolonged testosterone propionate administration on immature and adult female rat, 1126
Genitourinary Tract: See also Genitals; Urinary Tract; and under organs of genitourinary tract
congenital absence of abdominal musculature; associated changes in genitourinary tract and in spinal cord, *339
Genther, I.: Metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
Germanin, Therapy: See Lupus erythematosus
Gerstenberger, H. J.: Phosphate solutions in treatment of infantile rickets, 668
Ginandes, G. J.: Insensible perspiration in children; influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71
Gingivitis: See Gums, inflammation
Glands: See Endocrine Glands; Lymph Nodes; and under names of individual glands
Glioma, familial, 436
of pons; clinical and pathologic characteristics, 1335
Globulin in Blood: See Blood, proteins
Placental: See under Placenta
Globus Pallidus: See Lenticular Nucleus
Glomerulonephritis: See under Nephritis
Glycemia: See Blood, sugar
Glycosuria: See Dextrose
Goats, Milk: See under Milk
Goethlin Test: See Capillaries, permeability
Goettsch, E.: Reaction of edematous patient to transfusion with serum or with acacia, 915
Goiter: See also Thyroid
effect of yeast and of thiamin on production of low iodine goiter, 641
exophthalmic, in children, 431
sporadic congenital goiter causing death of infant, *1067
Goldberger, E. W.: Old tuberculin and purified protein derivative in tuberculin patch test; comparative study, *527
Goldbloom, A.: Cardiac irregularities in infants, 1147
Gonadotropic Substances: See under Androgens; Hormones, sex; Pituitary Body; Pituitary Preparations; Pregnancy, urine in
Gonads: See also Endocrine Glands; Genitals; Hermaphroditism; Ovary; Testicles
Gonads—Continued
thymus studies of some changes in gonads and pituitary following its destruction by roentgen irradiation, 890
Gonococci, Infections: See under Gonorrhea
Gonorrhea, favorable action of sulfanilamide (1162 F) associated with local treatment in gonococcic vulvovaginitis in little girls, 639
gonococcal peritonitis, 410
treatment of gonococcic vaginitis in infancy with sulfanilamide, 639
Grants, Elizabeth McCormick grant awarded, 1371
Granulocytopenia associated with sulfanilamide therapy, 224
neutropenia following sulfanilamide; report of case, 224
Graves' Disease: See Goiter, exophthalmic
Greenfield, J.: Acute suppurative thyroiditis during childhood, *837
Greenspan, L.: Exanthema subitum (roseola infantum), *983
Greenstein, N. M.: Hourglass tumors of spine in children; review of literature and report of case, *131
Grip: See Influenza
Growth: See also Diet and Dietetics; Nutrition; etc.
and development of newborn infants; factors associated with ossification of center for cuboid bone, 678
and physical development in mongolism, 213
biometric study in premature infant, 1101
body measurements of Texas school children, 490
effect of injections of extract of thymus (Hanson) on growth of albino rat, 1342
effect on growth rate of thyroparathyroidectomy in newborn rats and of subsequent administration of thyroid, parathyroid and anterior hypophysis, 640
effects of thymectomy on growth of albino rats, 1342
experimental phases of pineal problem, 1127
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on calcium retention, *1186
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on nitrogen retention, *965
obesity in childhood; physical growth and development of obese children, *457
treatment of osteomyelitis during period of growth, 891
vitamin E; its influence on lactation of mothers and growth of their children, 855
Grulee, C. G.: Newborn; president's address, *1
Günther Syndrome: See Polycythemia
Guest, G. M.: Role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Guillain-Barré Syndrome: See Nerves, roots
Gums, fusospirillar stomatitis, 1117
inflammation; investigations of vitamin C standard in healthy children and in children suffering from gingivitis, 398
Gunn Phenomenon: See Jaw-Winking Phenomenon
Gynecology, gynecologic problems of adolescent girl, *162
Gynecomastia: See Breast, hypertrophy
György, P.: Hepatic injury on nutritional basis in rats, 920

Habit formation, 444
Haemophilus Pertussis: See Whooping Cough
Hair, color of; white forelock; new mutation, 1132

Hair—Continued
excessive; hydroa aestivale in congenital porphyria, 1132
Hamilton, W. F.: Accuracy of clinical determinations of blood pressure in children with values under normal and abnormal conditions, *102
Hand: See also Fingers and Toes
congenital abnormalities of extremities, 1100
identity of 2 hands in 1 sister and extreme dissimilarity of 2 members in another, 1349
type of osseous dystrophy affecting hands and feet, 218
Hand, A.: Two cases of complete recovery from sporadic cretinism, 910
Hand's Disease: See Schüller-Christian Syndrome
Harelip, hyperpyrexia following operation for cleft lip or palate, *320
repair of, 224
Hartwell, R. M.: Meningococcic endocarditis and myocarditis, *823
Haskin, G. E.: Active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
Havens, F. Z.: Severe hemorrhage secondary to retropharyngeal abscess, *1256
Hawke, W. A.: Preliminary observations on dilantin therapy of convulsions, 1143
Hay Fever: See also Anaphylaxis and Allergy
nasal allergy, 220
Head: See Cranium
Hearing: See Deafness
Heart: See also Arrhythmia; Cardiovascular System; Endocarditis; Pericarditis; and under names of various conditions, as Diphtheria; Rheumatic Fever; etc.
abnormalities; complete transposition of great vessels, 1325
abnormalities; congenital cardiac pentad; Fallot's so-called tetralogy together with common interventricular orifice, 199
abnormalities; congenital cardiopathy without murmurs, 1327
abnormalities; diagnosis of congenital cardiac defects in infancy; study of 32 cases with necropsies, *8
abnormalities; 4 cases of congenital cardiac disease, 1112
abnormalities; nephritis with albuminuric retinitis and patent ductus arteriosus, 638
anomalous drainage of pulmonary veins into coronary sinus, *354
block, importance in newborn, 403
block in scarlet fever, 1317
Böttner and Kauffmann method of determining heart function, 1112
cardiac irregularities in infants, 1147
circulatory causes of cyanosis; panel discussion, 1310
circulatory function in anemias of children; roentgenographic measurement of cardiac size, *250
disease, multiple pregnancies in woman with congenital cyanosis due to, 1326
disease; radiologic aids in diagnosis in children; review, 415
disease; studies on volume of circulating blood in children with heart disease, 872
displacement; air cysts of left lung with displacement of heart to right in infant, 1116
electrocardiogram taken from wall of normal chest in childhood, 416
electrocardiography, value in diphtheria, 1315
fetal heart sounds studied by phonocardiograph, 872
human fetal electrocardiogram, 184
Heart—Continued
hypertrophy; Klippel-Feil syndrome with cardiac hypertrophy in newborn infant, 186
inflammation; sudden death of boy of 12 years, 622
influence of diphtheria on measurements of heart and alterations of heart muscle tissue, 408
investigation concerning QT interval of electrocardiogram in diphtheria of children, 416
meningococcic endocarditis and myocarditis, *823
myocardial changes in diphtheria, 863
paroxysmal tachycardia, 1112
scarlet fever heart, 1316
sounds in normal and pathological conditions, 200
Valves: See under names of valves
when shall digitalis be given in diphtheria? 1315
Heat: See also Temperature
comparative study of metabolism of certain aminoacids with reference to respiratory exchange and heat production, 848
Production: See Metabolism
Height: See under Body
Heine-Medin Disease: See Poliomyelitis
Helminths and Helminthiasis: See Intestines, parasites
Hemangioma: See Angioma
Hematocolpos: See under Vagina
Hematocrit: See Erythrocytes
Hematoma: See also Meninges, hemorrhage
enormous hematoma of scalp resulting in death, 1311
Hematoporphyria, hydroa aestivale in congenital porphyria, 1132
Hemoglobin and Hemoglobin Compounds: See also Anemia; Blood; Erythrocytes; Hemoglobinuria; etc.
physiological effects of sulfanilamide; methemoglobin formation and its control, 1348
Hemoglobinuria, paroxysmal myopathic crises with hemoglobinuria, 645
Hemophilia: See also Diathesis, hemorrhagic
sex ratios in families with hemophilia, *1215
Hemopoietic System: See also under Blood; Erythrocytes
clinical studies of behavior of reticulocyte count, 873
clinical study of reticulocyte count in blood diseases of children, 873
Diseases: See also Anemia; Leukemia; etc.
hydrops foetalis (erythroblastosis), 607
Hemoptysis, death from hemoptysis occurring in infant of 9 months, 1322
Hemorrhage: See also Blood, coagulation; Encephalitis, hemorrhagic; Hemophilia; Purpura, haemorrhagica; and under names of organs and regions, as Adrenals; Brain; Intestines; Meninges; Retina; etc.
pneumonia complicated by acute hemorrhage and ulcerative gastroenteritis in child, *1265
severe, secondary to retropharyngeal abscess, *1256
Henoch's Purpura: See Purpura
Heparin, normal antithrombin of blood and its relation to heparin, 1325
Hepatitis: See Jaundice; Liver
Heredity, congenital disease, 402
Hermaphroditism, pseudohermaphroditism, 216
pseudohermaphroditism, adiposity, polyuria and hyperglycemia; infundibulotuberian syndrome, 630
Hernia, diaphragmatic, 628, 880

Hernia—Continued
diaphragmatic; lung diseases; pulmonary tuberculosis, mild bronchiectasis, staphylococcal empyema and diaphragmatic hernia into thorax, 420
in infancy, 1117
right diaphragmatic hernia, 422
Herpes, herpetic stomatitis; enanthem, 907
Herter-Gee Disease: See Celiac Disease
Hibbs, W. G.: Agenesis of lung; review of literature and report of case, *571
Higgins, H. L.: Simpler methods for determining basal metabolic rates of children, 670
Hill, L. W.: Immunologic relations between cow's milk and goat's milk, 905
Hinton Test: See Syphilis
Hip Joint, Deformities: See Osteochondritis deformans
early diagnosis of congenital dislocation, 224
osteochondritis dissecans of, 217
Hirschsprung's Disease: See Colon, dilatation
Hirsutism: See Hair, excessive
Hodgkin's Disease: See Lymphogranuloma
Hoelscher, H.: Active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
Hoffmann-Werdnig Paralysis: See Atrophy, muscular
Hog-Cholera, Bacilli: See Salmonella suipestifer
Hogden, C.: Metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
Holland, D. F.: Disabling diseases of childhood; their characteristics and medical care as observed in 500,000 children in 83 cities canvassed in National Health Survey of 1935-1936, *1157
Holt, L. E., Jr.: Nutritional requirements in inanition, 669
Honey, comparative values of various carbohydrates used in infant feeding, 178
Hormones: See also Adrenal Preparations; Endocrine Glands; Insulin; and under names of organs, as Parathyroid; Pituitary Body; Thyroid
Lactogenic: See Pituitary Preparations
Sex: See also Androgens; Estrogens; Pituitary Preparations; Pregnancy, urine in; and under names of organs, as Ovary; Pituitary Body; etc.
sex; effect of antuitrin S on thymus of young albino rat, 641
Horton, J.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Hospitals, air contamination and air sterilization, *1023
bringing up and teaching of children in institutions for sick, 427
Huepel, R. C.: Familial cleidocranial dysostosis (cleidal dysostosis); preosseous and dentinal dystrophy, *786
Huet-Pelger's Nuclear Anomaly: See Leukocytes, count
Humidity, effects of high humidity on skin temperature at cool and warm conditions, 1348
Hummel, F. C.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Hunger, effect of alcohol on hunger sense, 627
nutritional requirements in inanition, 669
observations on blood sugar level before, during and after hunger periods in humans, 627
occurrence of eosinophils in short periods of starvation, 1304
vitamin C in spinal fluid during hunger, 603
Hunscher, H. A.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Hurler Syndrome: See Bones, atrophy
Hydatid Cyst: See Echinococcosis
Hydramnion: See Amnion, dropsy of
Hydroa aestivale in congenital porphyria, 1132
Hydrocephalus, two hundred lumbar punctures of single patient, 1335
Hydrophobia: See Rabies
Hydrops Foetalis: See Fetus, pathology
Hymen, hematocolpos with congenital imperforate hymen, 1124
imperforate, and diagnosis of hematocolpos, 1341
Hypercholesteremia: See Blood, cholesterol
Hyperglycemia: See Blood, sugar; Diabetes Mellitus
Hyperkeratosis: See Keratosis
Hyperparathyroidism: See under Parathyroid
Hyperpyrexia: See Fever
Hypertelorism of Greig, 858
Hypertension: See Blood pressure, high
Hyperthyroidism: See under Goiter; Thyroid
Hypertrophy: See also under names of organs and regions, as Breast; Face; Heart; Thymus; etc.
unilateral; congenital hypertrophy of right side of body and left side of face, 184
Hypocholesteremia: See Blood, cholesterol
Hypogenitalism: See Genitals; Pituitary Body, diseases
Hypoglycemia: See Blood, sugar
Hypophysis: See Pituitary Body
Hypotension: See Blood pressure, low
Hypothalamus: See also Pituitary Body
histologic examination in ordinary obesity, in Morgagni's and Günther's syndromes and in morbus Cushing, 1120
pseudohermaphroditism, adiposity, polyuria and hyperglycemia; infundibulotuberian syndrome, 630
Hypothyroidism: See under Thyroid
Hysteria in childhood, 213
some hysterical syndromes at puberty and at prepubertal periods, 638

Ichthyosis, congenital, 1312
Icterus: See Jaundice
Idiocy: See also Feeblemindedness
amaurotic family idiocy, 1121
amaurotic; Tay-Sachs disease, 436
depth and nerve-cell content of supragranular cortex in normal and mentally defective persons, 426
juvenile amaurotic idiocy, 884
late forms of amaurotic idiocy showing picture of paralysis agitans, 1122
Mongolian: See Mongolism
with blindness due to congenital cataracts in 2 brothers, 646
Ileocecal Valve, congenital anomaly of valve of Bauhin and of appendix as cause of peritonitis in newly born, 1312
Imbecility: See Idiocy
Immunity: See also under Anaphylaxis and Allergy; Antigens and Antibodies; Diphtheria; Serum; Streptococci; etc.

Immunity—Continued
immunologic relations between cow's milk and goat's milk, 905
Impetigo, control of, *931
impetiginous stomatitis, 422
Inanition: See Hunger
Index, cumulated, of American Journal of Diseases of Children, 1091, 1372
Infant Feeding: See also Diet and Dietetics; Nutrition; Vitamins; and under names of various foods, as Milk; etc.
acidified whole milk as infants' food, 180
ascorbic acid requirements in early infancy, 182
changes brought about in qualitative composition of bacterial flora in infants' stools by citric acid milk, 396
comparative incidence of infections in breast fed and artificially fed infants, 1146
comparative study of complementary feedings in 1,182 newborn infants, 185
comparative values of various carbohydrates used in, 178
complemental feeding and care of skin in newborn, 607
complementary feeding of suckling infant, 179
considerations in infant nutrition, 1304
diet in health and disease; breast feeding, 179
early introduction of solid foods in infant diet, 1144
effect of various feedings on loss and gain of weight during newborn period, 912
is goat's milk harmful as infant's food? 395
mineral and water metabolism in early infancy and its relation to problem of artificial feeding, 1095
modern decline of breast feeding, 597
postlactation feeding, 1350
psychologic hygiene of, 1121
roasted soybean in, 851
sulfur and protein metabolism of naturally and artificially fed infants, 1305
when does one begin artificial feeding in Budapest? 598
Infant Mortality: See also Newborn Infants; Premature Infants
in Batavia for years 1935-1936, 1308
Infant Welfare: See also Child Welfare
organization and functioning of infant consultation stations in rural area, 402
Infantilism, Intestinal: See Celiac Disease
Pituitary: See Pituitary Body, disease
Renal: See under Dwarfism
syndrome of infantilism, congenital webbed neck and cubitus valgus, 856
Infants: See also Children; Infant Feeding; Infant Mortality; Infant Welfare; Newborn Infants; Premature Infants; etc.
care; consideration of present day newborn nursery practice, 398
care; psychology, baby health and child welfare, 213
complement in newborn children, in infants and in fetuses, 860
Diseases: See under names of various diseases
education in early infancy, 214
Growth: See Growth
nutrition; treatment of nuralings' atrophy with extracts of liver and hypophysis, 851
white blood cell picture during first year of life, especially with regard to its course, 418
Infection: See also Immunity; Septicemia; and under names of bacteria, organs and regions
comparative incidence in breast fed and artificially fed infants, 1146
sulfanilamide therapy, 226
Infectious Diseases: See Communicable Diseases; Diphtheria; Infection; Pneumococci; Typhoid; etc.
Influenza, production of passive immunity against influenza virus by introducing immune serums into respiratory tract, 653
Injections: See also Blood, transfusion; Enemata; etc.
intradermal, actual site in skin of, 651
Injuries: See under Newborn Infants; and under names of organs and regions
Insensible Perspiration: See Metabolism
Instruments: See Apparatus
Insulin: See also Pancreas
comparative studies on use of standardized protamine zinc and crystalline insulin, 919
experimental studies concerning pathology of insulin convulsions, 918
experimental study of cerebral changes in prolonged hypoglycemia, 918
influence of lecithin and of lutein on hypoglycemic action of insulin, 642
lipodystrophy and lipomatosis from repeated injections of insulin, 1341
local lipomatosis caused by insulin injections, 1343
oral administration of hormonal proteins, 888
Therapy: See under Diabetes Mellitus; Pancreas
vitamin B fractions and insulin tolerance in albino rat, 1305
Intestines: See also Cecum; Colon; Gastrointestinal Tract; Rectum; Sigmoid
diseases; agar milk in dyspepsia and enterocolitis, 1334
enterogenous intramural cysts, *485
experimental studies of carbohydrate metabolism in, 181
gross hemorrhage due to pathologic condition in Meckel's diverticulum in 2 infants under 1 year of age, 1354
Intussusception: See Intussusception
Parasites: See also Amebiasis; Ascariasis; Dysentery
parasites; acute lymphocytic meningitis very probably verminous, 883
parasites; loss of weight caused by protozoa, 1319
parasites, statistical study in 1937, 618
parasites, survey in Chinese hospital patients in Shanghai, 413
prolapsus intussuscepti, 1116
Salmonella enteritis in infants, 423
sarcoma in children, 879
Intoxication: See under Digestive Tract; Toxemia; etc.
Intradermal Injections: See under Injections
Intussusception, 1117
acute, in children, 421
acute, in infancy treated by resection of bowel, 210
acute intestinal, in infants, 210
chronic, in children; 2 cases, 1333
jejunal, of newborn, *558
prolapsus intussuscepti, 1116
Iob, V.: Influence of cobalt on iron transportation in blood, 671
Sulfur metabolism of early infancy, *37
Iodide: See under Iodine and Iodine Compounds
Iodine and Iodine Compounds, certain iodine-reducing substances of thymus extract, 889
distribution and content of iodide in iodized salt obtained on open market throughout Michigan, 1370
role of iodine (potassium iodide) in experimental hyperthyreoidism, 642
Iris, absence of; aniridia congenita; irideremia, 893
ocular and dental malformations and disturbances of personality in 2 brothers with hereditary syphilis, 869
Irish, H. E.: Hyperpyrexia following operation for cleft lip or palate, *320

Iron deficiency anemia in children, *811
nutritional anemia in industrial district, 871
role of copper in iron-deficiency anemia in infancy, 620
Therapy: See Anemia; Trichocephaliasis
Irradiated Milk: See under Milk
Islands of Langerhans: See Pancreas

von Jaksch's Disease: See Pseudoleukemia, infantile
Jaundice, anemia of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
cerebral sequelae, after neonatal jaundice, ascribed to kernicterus, 608
epidemic icterus, 1334
familial icterus gravis of newborn, 404
icterus gravis with eclampsia in child during first year of life, 629
value of combined study of newer laboratory tests in differential diagnosis of toxic and obstructive jaundice including blood phosphatase, cholesterol partition, galactose tolerance and glucose tolerance, 208
Jaw-Winking Phenomenon, operation in case of Marcus Gunn phenomenon, 894
Jejunum, Intussusception: See Intussusception
Jenss, R. M.: Growth and development of newborn infants; factors associated with ossification of center for cuboid bone, 678
Jodar, E. O.: Tanning from ultraviolet irradiation brought out by blood transfusion, *1047
Johnson, V. C.: Thymus and thymicolymphatic constitution; roentgen findings, 1360
Johnston, A. T.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Johnston, J. A.: Continuous calcium and nitrogen balances before and after menarche, 672
Factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on calcium retention, *1186
Factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on nitrogen retention, *965
Johnston, L.: Herpetic stomatitis; enanthem, 907
Joints: See also under names of individual joints, as Hip Joint; etc.
Diseases: See also Arthritis; Arthritis deformans; Rheumatism; Tabes dorsalis; etc.
diseases; scorbutic subperiosteal hemorrhage complicated by osteomyelitis and pyarthrosis, 1095
hereditary arthrodysplasia associated with dystrophy of nails, 433
Josephs, H. W.: Studies of vitamin A, 665

Kajdi, C. N.: Nutritional requirements in inanition, 669
Kala-Azar: See Leishmaniasis
Kamochin: See under Bronchiectasis
Kaolin: See Dysentery
Kato, K.: Influence of cobalt on iron transportation in blood, 671
Katsampes, C.: Effect of hyperimmune human serum (lyophile) on humoral antibody titer in pertussis, *1234
Kauffmann Test: See Diuresis and Diuretics
Keith, J. D.: Metabolism of lactic acid in acute rheumatic fever, 1138
Kellman, L.: Primary polycythemia with leukemic manifestations, *146
Kelsey, W. M., Jr.: Congenital leukemia, *1268
Kempf, A. H.: Distribution of pneumococci in experimentally infected rats and penetration of antiserum into lesions, 1355
Keratoconjunctivitis, fate of patients suffering from phlyctenular disease with reference to tuberculosis, 437
Keratosis, cutaneous lesions due to vitamin A deficiency in children, 602
vitamin A deficiency in man; resolution of cutaneous lesions following parenteral administration of carotene, 183
Kernicterus: See under Jaundice
Ketosis: See Acidosis
Kidneys: See also Genitourinary Tract; Urinary Tract
atresia of ureter and agenesis of kidney, 1124
blood supply; eclampsia-like syndrome occurring in pregnant dogs and rabbits following renal artery constriction, 1339
Diseases: See also under Nephritis
diseases; anasarca due to nephrosis and intercurrent erysipeloid cutaneous lesions, 640
diseases; assimilation of protein by young children with nephrotic syndrome; effect of dietary fat and carbohydrate on nitrogen balance, *935
diseases; assimilation of protein by young children with nephrotic syndrome; effect of nephrotic crises on assimilation of nitrogen, *939
diseases; concentrated human blood serum as diuretic in nephrosis; further observations, 914
diseases; glomerulotubular nephropathy in Schoenlein-Henoch purpura, 416
diseases; metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
diseases; nephropathies in course of acute nasopharyngeal diseases of infants, 1340
function, direct evidence in early human fetus, 638
renal adenosarcoma and subcutaneous elimination pyelography, 640
renal lesion in hyperparathyroidism, 1127
review of differential diagnosis of osteopathies due to hormonal causes, avitaminosis and kidney disease, 1345
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
rupture of, 1340
rupture of congenital solitary kidney, 1123
syndrome consisting of affections of kidney, stunted growth, rickets and disturbed cystine metabolism, 215
tumors; Wilms' embryoma, 1124
tumors; Wilms' tumors, 428
Killingsworth, W. P.: Agenesis of lung; review of literature and report of case, *571
Klippel-Feil Syndrome: See Spine, abnormalities
Koch's Bacillus: See Tubercle Bacilli
Koller, L. R.: Air contamination and air sterilization, *1023
Koplik's Spots: See Measles, diagnosis
Kupffer Cells: See under Liver
Kyphosis: See Spine, curvature

Labor: See also Obstetrics; Placenta; Pregnancy; etc.
Complications: See Cesarean Section; Fetus; Newborn Infants; etc.
constitutional factors in etiology of prematurity, 1101
fying fetus, 857
placental interchange; comparison of total base concentration of fetal and maternal blood at parturition, 858
Laboratories, advice on regulations of laboratory technic, 652
Lacrimal Organs, treatment of congenital atresia of nasolacrimal duct, 647

Lactation: See also Colostrum; Infant Feeding; Milk
clinical and histologic investigations of so-called witch's milk lactation with attention to its relation to birth weight of infant, 1312
hemosedimentation of healthy lactating women, 1328
inhibition during puerperium by testosterone propionate, 641
lactogenic hormone, 1126
postlactation feeding, 1350
vitamin E; its influence on lactation of mothers and growth of their children, 855
Lactose, comparative values of various carbohydrates used in infant feeding, 178
Langerhans' Islands: See under Pancreas
Langford, W. S.: Thumb and finger sucking in childhood, *1290
Language, how Hungarian girl aged 7 learned French, 1122
Laryngitis: See under Larynx
Laryngotracheobronchitis: See Respiratory Tract
Larynx, acute laryngitis in infants, 1328
congenital stridor, 625
Diphtheritic: See Diphtheria, laryngeal
laryngitis chronica hypertrophica in childhood, 874
stenosis, 625
stridor, 1359
stridor congenitus, 1329
Laurence-Biedl Syndrome, Fröhlich syndrome; report of original case, *1282
medulloblastoma associated with adiposogenital syndrome, 884
Lawrence, B. A.: Pneumonia complicated by acute hemorrhage and ulcerative gastroenteritis in child, *1265
Lead poisoning, clinical and radiologic studies, 443
poisoning; lead colic, 442
poisoning produced by irradiated lead, 443
Lederer's Anemia: See Anemia, hemolytic
Legg's Disease: See Osteochondritis deformans juvenilis
Legs, Gangrene: See Gangrene
Leishmaniasis, diagnosis of kala-azar by search for parasite in bone marrow (puncture of metaphysial region of tibia), 411
diagnosis of kala-azar by sternal puncture, 1319
sternal puncture technic and its clinical value with reference to its usefulness in diagnosis of kala-azar, 1108
Lens, Crystalline: See also Cataract
dislocation of ocular lenses of premature infant; dolichostenomelia? 646
Lenticular Nucleus, hepatolenticular degeneration following trauma, 632
Leukemia, acute (hemocytoblastic), 1327
aleukemic myelosis with skeletal changes in 7 year old boy, 1112
blood iodine in, 1111
congenital, *1268
congenital, with "chloroma," *332
hypertrophy of thymus in course of acute leukemias of childhood, 1326
myelogenous chloroma, 1328
primary polycythemia with leukemic manifestations, *146
Leukocytes: See also under Blood; Eosinophils; Leukemia; Mononucleosis; etc.
count; further research and observations on familial deviations of nuclei of leukocytes (Pelger-Huet), 623
count; local and peripheral leukocyte counts in certain infectious diseases, 1315
count; relation of leukocytosis to ascorbic acid requirements, 852
Leukocytes—Continued
count; total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
count; total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
occurrence of eosinophils in short periods of starvation, 1304
white blood cell picture during first year of life, especially with regard to its course, 418
Leukocytosis: See Leukocytes, count
Leukodystrophy: See Brain, diseases
Leukopenia: See Granulocytopenia; Leukocytes, count
Levine, M. I.: Sequence of roentgen evidence of tuberculosis and cutaneous sensitivity to tuberculin; study of infants, *799
Levulose, comparative values of various carbohydrates used in infant feeding, 178
Levy, W.: Acute hemolytic anemia; Lederer type, *349
Lewis, J. H.: Jejunal intussusception of newborn, *558
Leyden-Moebius' Disease: See Dystrophy, muscular
Lichtenstein, B. W.: Congenital absence of abdominal musculature; associated changes in genitourinary tract and in spinal cord, *339
Life expectancy after ureteral transplantation, 428
Light, Therapy: See Ultraviolet Rays; and under names of diseases
Limbs: See Extremities
Lindberg, R.: Preparation of mold antigens for diagnostic and therapeutic purposes, 1367
Lipids: See under Lipoids
Lipodystrophy: See Fat
Lipogranulomatosis, subcutaneous adiponecrosis and lipogranulomatosis in infant, 222
Lipoids: See also Cholesterol; Fat; Phosphorus and Phosphorus Compounds
clinical and experimental studies of lipoid pneumonia, 654
Lipomatosis: See Fat
Lips, Diphtheria: See Diphtheria
Harelip: See Harelip
Listerella Monocytogenes: See Bacteria, monocytogenes
Lithiasis: See under Gallbladder
Little's Disease: See Paralysis, spastic
Livedo telangiectatica congenita generalisata, problem of, 441
Liver, abscess; pyogenic abscess of liver in 9 year old child, 1332
correlation of cecum and liver in clinical conditions in children, 629
cultivation of vaccinia virus with reticuloendothelial cells (Kupffer cells), 610
diet and rate of depletion of hepatic vitamin A, 1096
diseases; generalized edema with hypoproteinemia, 180
Extracts: See also under Diarrhea
extracts, preparation and properties of charcoal adsorbate of, 1325
extracts; treatment of nuralings' atrophy with extracts of liver and hypophysis, 851
familial juvenile cirrhosis, 1334
familial juvenile cirrhosis; clinical study, 1333
function tests; value of combined study of newer laboratory tests in differential diagnosis of toxic and obstructive jaundice including blood phosphatase, cholesterol partition, galactose tolerance and glucose tolerance, 208

Liver—Continued
hepatic injury on nutritional basis in rats, 920
hepatolenticular degeneration following trauma, 632
new knowledge on relation of liver function to vitamin B_1, 855
Oils: See Cod Liver Oil; Oil
Therapy: See Diarrhea
Lobectomy: See under Bronchiectasis
Locomotion, disturbance of gait, probably following scarlatinal encephalitis, 1314
left upper abdominal pain on effort in juvenile rheumatic disease; new symptom of low grade active phase, 865
Löffler Syndrome: See Eosinophilia; Lungs, pathology; Tuberculosis, pulmonary
Lordosis: See Spine, curvature
Lues: See Syphilis
Lumbar Puncture: See Spinal Puncture
Lungs: See also Pleura; Respiration; Respiratory Tract; Thorax; etc.
abnormalities, congenital, and acquired bronchiectasis, 878
abscess; multilocuated abscess of lung in infant, cured by treatment with intravenous injections of alcohol, 626
absence, congenital, of left lung, 205
agenesis and hypoplasia of, 878
agenesis of; review of literature and report of case, *571
air cysts of left lung with displacement of heart to right in infant, 1116
Collapse: See also Pneumothorax
collapse; right middle lobe collapse, 206
collapse; role of allergy in atelectasis in children, *237
congenital air cysts in infants, 878
congenital cystic disease, 205, 206
Diseases: See Tuberculosis, pulmonary; etc.
erroneous diagnosis of bullous emphysema of child's lung, 1331
hydatid cysts of, 1331
marconitherapy in nontuberculous pulmonary diseases, 627
nonspecific infections in childhood, 877
pathology; syndrome of Löffler, 878
pseudomiliary lesions in, 627
pulmonary causes of cyanosis; panel discussion, 1310
Surgery: See under Bronchiectasis
Tuberculosis: See Tuberculosis, pulmonary
Vital Capacity: See Vital Capacity
Lupus erythematosus, Bayer 205 (germanin) in treatment, 438
Lutein: See Ovary, hormones
Lye poisoning and stricture of esophagus, 1117
poisoning of children in Slovakia, 227
treatment of caustic (lye) poisoning in children's clinic of University of Budapest, 652
Lymph Nodes: See also Lymphogranuloma; etc.
development of tuberculosis of cervical lymph nodes, 619
effect of prepuberal castration of female albino rat on thymus and lymph glands, 1342
suppurative streptococcic lymphadenitis following diphtheria in brothers, 864
Lymphangioma: See Angioma
Lymphatic System: See also Lymph Nodes
thymus and thymicolymphatic constitution; clinical aspects as seen by obstetrician, 1358
thymus and thymicolymphatic constitution; clinical aspects as seen by otolaryngologist, 1359
thymus and thymicolymphatic constitution; introductory remarks, 1357
thymus and thymicolymphatic constitution; roentgen findings, 1360
Lymphatism: See Thymus, hypertrophy
Lymphocytes: See Leukemia; Leukocytes
in Meningitis: See under Meningitis
Lymphogranuloma, blood iodine in leukemia, 1111
Schaumann's "benign granulomatosis" (morbus Besnier-Boeck-Schaumann), 226
Lyon, G. M.: Epidemic diarrhea of newborn, 662
Lyon, R. A.: Causes of prematurity; review of literature, *586
Studies in immunity; spontaneous changes in Dick and Schick reactions of children, *734
Lyon, W.: Strychnine poisoning in 16 month old infant, 1368
Lyttle, J. D.: Reaction of edematous patient to transfusion with serum or with acacia, 915

M & B 693: See Endocarditis; Sulfanilamide and Sulfanilamide Derivatives
McClelland, J. E.: Active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
McCormick, Elizabeth McCormick grant awarded, 1371
McCreary, J. F.: Treatment of chronic eczema in children, 1145
McCune, D. J.: Late rickets with glycosuria and organic acid acidosis (Fanconi type), 673
McGuinness, A. C.: Effect of hyperimmune human serum (lyophile) on humoral antibody titer in pertussis, *1234
Macklin, M. T.: Sex ratios in families with hemophilia, *1215
MacLaren, W. R.: Simpler methods for determining basal metabolic rates of children, 670
MacNair, V.: Effect of dietary supplement on ossification of bones of wrist in institutional children; effect of cod liver oil supplement, *295
McQuarrie, I.: Experimental studies concerning pathology of insulin convulsions, 918
Macrogenitosomia: See under Adolescence
Macy, I. G.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Madelung Deformity: See Wrist, deformities
Magnesium sulfate; avenues of absorption of magnesium sulfate and its clinical application, 1349
Sulfate, Therapy: See Chorea; Tetanus
Malaria, accidental transmission through injection of whole blood, 192
blackwater fever following administration of "atebrin," 1318
chemoprophylaxis in Portuguese India, 867
demonstration of sporozoites in human tissues, 410
diagnosis of azar, 412
experimental studies on treatment of malarial splenomegalies by method of Ascoli, 1108
in infant 7 months old following injections of paternal blood, 193
in Panama; eighth year's observation, 410
serologic reactions in syphilis and malaria in infancy, 617
some facts about congenital malaria, 1108
tertian, sulfanilamides in, 192
use of atabrine as prophylactic agent; 2 years' observation, 409
Malformations: See Abnormalities and Deformities; Monsters; and under names of organs and regions
Mallory, E. W.: Sequelae of postinfectional encephalitis with reference to mental involvement, 1352

Malnutrition: See under Dystrophy; Nutrition
Mammary Gland: See Breast
Mantoux Test: See under Tuberculosis
Mapharsen: See Syphilis
Marasmus: See Nutrition
Marfan Syndrome: See Fingers and Toes, abnormalities
Maroney, J. W.: Factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on calcium retention, *1186
Factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on nitrogen retention, *965
Mason, H. H.: Late rickets with glycosuria and organic acid acidosis (Fanconi type), 673
Massalski Method: See Tuberculosis
Mastoiditis: See also Otitis Media
para-aminobenzenesulfonamide and its derivatives in treatment of beta hemolytic streptococcus infections of middle ear and mastoid; report of 6 cases in children, 202
sulfanilamide in treatment of acute mastoiditis, 1113
without middle ear involvement; 4 cases, 418
Maternity: See also Labor; Obstetrics; Pregnancy
welfare; maternal and child health services under Social Security Act, 1100
welfare, maternal and child health under National Health Program, 903
Measles, case of scleredema following, 439
conduct of school epidemic, 407
cord blood serum for prophylaxis and modification of, 1357
diagnosis; suggestion for earlier recognition of Koplik's spots, 404
German: See Rubella
local and peripheral leukocyte counts in certain infectious diseases, 1315
placental globulin in modification of, 187
preeruptive, treatment with convalescent serum, 405
prophylactic and therapeutic value of placental extract in regard to measles, scarlatina and mumps, 190
prophylaxis against, 190
reticulocytes in, 409
use of placental extract in epidemic, 407
Meat, distribution of vitamin B_1 (thiamin) in meat and meat products, 1096
Meckel's Diverticulum: See under Intestines
Mediastinum, pneumographic examination of anterior mediastinum in diagnosis of atypical forms of hypertrophy of thymus, 891
tumors in children, 207
Medicine, preventive, some limitations in, 1347
Medin-Heine Disease: See Poliomyelitis
Medulloblastoma associated with adiposogenital syndrome, 884
Megacolon: See Colon, dilatation
Melanin, pigmentation; melanosis coli in boy aged 2½ years, 208
Melanosis: See Melanin, pigmentation
Mellencamp, F. J.: Hypothyroidism in children, 1353
Menarche: See Menstruation
Meninges, hemorrhage of newborn, 859
hemorrhage; relapsing juvenile chronic subdural hematoma; clinical and roentgenographic study, 210
intracranial tuberculoma, 212
retinal hemorrhages in association with meningeal hemorrhage of newborn, 1103
spinal fluid in normal newborn infants and infants with meningeal hemorrhage, 608
spontaneous subarachnoid hemorrhage in children, 425
Meningitis: See also Arachnoiditis; Meningococci; Meningoencephalitis
acute lymphocytic meningitis, 1335
Meningitis—Continued
acute lymphocytic meningitis very probably verminous, 883
beta hemolytic streptococcus meningitis, 881
caused by pneumococcus type III; observations on sulfanilamide therapy, *274
cerebrospinal, serum therapy and new factors in treatment, 613
danger of primary abdominal tuberculosis in children, 618
differentiation between meningitis and encephalitis by color reaction of spinal fluid, 1337
futility and danger of intrathecal mercurochrome in, 881
immunological aspects and treatment, 881
meningeal reactions to serum, 1106
meningeal symptoms caused by Ascaris, 1321
meningococcal, sulfanilamide therapy; report on 3 cases, 406
meningococcal, treatment with M. & B. 693, 407
meningococcal, treated with proseptasine and soluseptasine, 1106
meningococcic, 861
meningococcic sepsis running course of erythema exsudativum multiforme with subsequent meningitis, 864
otitic, due to Streptococcus hemolyticus; operation and recovery, 630
pediatric treatment of otologic sepsis, including meningitis, 1113
pneumococcic, of newborn, 403
pneumococcic, treatment of, 900
problems of suppurative petrositis and its surgical treatment together with outline of prophylaxis and treatment of otitic meningitis, 203
purulent, cured by sulfanilamide; discussion on nature and possible relapse of streptococcic meningitis, 627
purulent, due to meningococcus in infant, cured by combined serotherapy and chemotherapy, 613
response to antiserums in meningococcic infections of human beings and mice; comparative study, *746
streptococcic, intravenous injection of hypotonic salt solution containing sulfanilamide, 899
streptococcic, originating in mastoid cured by oral and intrasphenoid injections of sulfanilamide, 881
sulfanilamide in treatment of acute infections of central nervous system, 424
sulfanilamide in treatment of brain abscess and prevention of meningitis, 899
symposium; what is justifiable to do in otitic meningitis; brief (historical) review of operative treatment of purulent meningitis; surgical indications in suppurative meningitis; surgical treatment of meningitis; sulfanilamide and related compounds in treatment of meningitis secondary to ear and sinus infections, 623
treatment by means of sulfanilamide, 1120
treatment of acute forms with sulfanilamide, 882
treatment of purulent streptococcic meningitis by sulfanilamide, 631
tuberculous; cerebral paratuberculosis, 413
tuberculous, pathologic nature, 620
tuberculous, some methods for diagnosis of, 1109
Meningococci: See also under Meningitis
meningococcal broth culture filtrates; failure of protection experiments, 1105
meningococcic endocarditis and myocarditis, *823
meningococcus and meningococcic infection, 1147

Meningococci—Continued
response to antiserums in meningococcic infections of human beings and mice; comparative study, *746
sulfanilamide in treatment of 106 patients with meningococcic infections, 404
Meningoencephalitis: See also Encephalitis; Meningitis
due to Cryptococcus meningitidis (Torula histolytica), 1119
Menstruation, continuous calcium and nitrogen balances before and after menarche, 672
incompatibility of mother's milk obtained during menstruation in infant with exudative diathesis, 1133
Mental Diseases: See Feeblemindedness; Nervous and Mental Disabilities; etc.
Mental Tests, criticism in mental testing, 637
mentality of dispensary epileptic patients, 213
Mercurochrome, Therapy: See Meningitis
Mercury: See also Diuresis and Diuretics
mercurial poisoning from bichloride of mercury enema, 651
Vapor Lamp: See Ultraviolet Rays
Merzbacher-Pelizaeus' Disease: See Brain, diseases
Metabolism: See also under specific headings, as Carbohydrates; Fat; Minerals; Nitrogen; etc.
basal, of tuberculous children; afebrile primary pulmonary tuberculosis, *119
basal, of tuberculous children; children with pneumothorax, *957
basal, of tuberculous children; tuberculosis of bone, *778
calcium requirements of 5 preschool girls, 1094
calcium requirements of infants and children, 1094
comparative study of metabolism of certain aminoacids with reference to respiratory exchange and heat production, 848
effect of sleep on insensible perspiration in infants and children, *82
energy content of goat milk, 1094
in experimental diphtheria of dog, 408
insensible perspiration in children; influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71
new creatinine standard for basal metabolism and its clinical application, *506
obesity in childhood; basal metabolism and serum cholesterol of obese children, *1001
simpler methods for determining basal metabolic rates of children, 670
Meteorology, relation between sunspots and diphtheria, 1316
Methemoglobin: See Hemoglobin and Hemoglobin Compounds
Methylthionine Chloride, physiological effects of sulfanilamide; methemoglobin formation and its control, 1348
Micturition: See Urination
Migraine, ocular defects as cause in children, 220
present day status of therapy, 211
Milk: See also Infant Feeding
acidified whole milk as infants' food, 180
bacterial growth and lactic acid production in citric acid milk, 395
bacteriological observations on acid milk in inner Mongolia, 599
changes brought about in qualitative composition of bacterial flora in infants' stools by citric acid milk, 396
citric acid fermentation by bacteria, 396
copper content in anemic infants, 181
Goat's: See also under Anemia
goat's, energy content of, 1094
goat's; is goat's milk harmful as infant's food? 395
Milk—Continued
Human: See also Infant Feeding; Lactation
human, copper content of, 180
human; incompatibility of mother's milk obtained during menstruation in infant with exudative diathesis, 1133
human; mechanical extraction of breast milk, 851
immunologic relations between cow's milk and goat's milk, 905
influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
irradiated, effect on storage of nitrogen and acid-base minerals in children, *753
outbreak of milk-borne typhoid, 193
phosphorus esters in human, cow's and goat's milk, 180
reappraisal of vitamin D milks, 178
report on market supplies of certain urban communities, 179
sanitary control in Montreal, 1098
study of breed and seasonal variations in ascorbic acid content of certified milk from Guernseys and Holsteins, 1096
why do not more children get milk-borne infections? 860
Witches': See under Lactation
Miller, M. J.: Acute hemolytic anemia; Lederer type, *349
Miller, N. F.: Thymus and thymicolymphatic constitution; clinical aspects as seen by obstetrician, 1358
Minerals: See also Iron; etc.
and water metabolism in early infancy and its relation to problem of artificial feeding, 1095
effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Mitchell, A. G.: Incidence of dysentery in gastroenteritis, 665
Studies in immunity; spontaneous changes in Dick and Schick reactions of children, *734
Mitral Valve, congenital and acquired heart disease and its retrospective diagnosis, 200
Moebius-Leyden's Disease: See Dystrophy, muscular
Moeller-Barlow's Disease: See Scurvy
Molds: See Fungi
Molony, C. J.: Role of allergy in atelectasis in children, *237
Mongolism, clinical, in Negro, 1338
congenital megacolon in child with mongolism, 629
dysphonia aspects of mongolian imbecility, 1338
etiologic study of, 1338
function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
growth and physical development in, 213
mongolian idiocy, 638
studies in; thyroid gland, 1338
Monocytes: See Leukocytes; Mononucleosis; etc.
Mononucleosis, infectious, isolation of Listerella monocytogenes from, 1302
Monsters, duplication of hind-end of anencephalic fetus, 857
monstrosity following birth of 9 normal children, 856

Monypenny, D.: Early introduction of solid foods in infant diet, 1144
Moon-Laurence-Biedl Syndrome: See Laurence-Biedl Syndrome
Moore, E.: Immunologic properties of scarlatina convalescent serum, *1039; correction, 1372
Morbus Cushing: See Pituitary Body, diseases
Maculosus: See Purpura haemorrhagica
Morgagni Syndrome: See Cranium, pathology
Morphine, toxicity in newborn rat, 185
Morrison, M.: Congenital leukemia with "chloroma," *332
Morse, M.: Physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
Mortality Statistics: See Infant Mortality; Newborn infants; Vital Statistics; etc.
Mouth: See Angina, Vincent's; Gums; Stomatitis; etc.
Mulligan, F.: Comparative incidence of infections in breast fed and artificially fed infants, 1146
Mumps: See Parotitis
Murmurs: See Heart, murmurs
Muscles, Atrophy: See Atrophy, muscular
congenital absence of abdominal musculature; associated changes in genitourinary tract and in spinal cord, *339
Dystrophy: See Dystrophy, muscular
lactic acid formation by muscles of scorbutic guinea pigs, 1343
muscular hypertonicity in infants of nutritional cause, 179
primary tuberculosis of, 1323
roentgen studies of patterns of growth of muscle and other tissues in young children, 680
Myasthenia gravis; tumors of thymus and their association with myasthenia, 642
Myatonia, amyotonia congenita (Oppenheim), 217
congenita, clinical aspects of, 1345
congenita in identical twins, 435
Myelitis: See Encephalomyelitis
Myelosarcoma simulating chloroma, 1112
Myelosis, Aleukemic: See Leukemia
Erythremic: See Pseudoleukemia, infantile
Myers, J. A.: Tuberculosis in children less than 6 years of age, *92
Myocarditis: See Heart
Myocardium: See Heart
Myoclonus, congenital heredofamilial disease, consisting of rhythmic tremor of head, eyes and upper extremities, 631
Myositis: See also Dermatomyositis
fibrosa, interstitial, in girl 7 years of age, 1345
Myotonia, congenita, quinine in, 433
in child; improvement by quinine treatment, 217
Myxedema: See also Thyroid, hypothyroidism
calorigenic potency of free thyroxine by mouth, 1125

Nails, hereditary arthrodysplasia associated with dystrophy of nails, 433
Nasolacrimal Duct: See Lacrimal Organs
Nasopharynx, method for concentration of poliomyelitis virus in nasopharyngeal washings, 609
nephropathies in course of acute nasopharyngeal diseases of infants, 1340
new method of obtaining sputum from infants and children thought to have pneumococci in nasopharynx, 1139
National Health Program, maternal and child health under, 903
Neck, pressure on nerves in neck, 1360
pressure on vessels of neck, 1360
syndrome of infantilism, congenital webbed neck and cubitus valgus, 856
Neck—Continued
teratoma of neck of fetus with acute hydramnios, 1308
Necrosis: See also under Bones; Fat; Kidneys; etc.
subcutaneous fat necrosis of newborn, 404
Nematodes: See Ascariasis; Oxyuriasis; etc.
Neoplasms: See Cancer; Sarcoma; Tumors; etc.
Nephritis: See also Albuminuria; Kidneys, diseases
acute, treatment in infants, 640
chronic, classification of, 216
nephrosis treated by Osman method (alkali therapy), 1123
nephrotic, 1124
with albuminuric retinitis and patent ductus arteriosus, 638
Nephrosis: See Kidneys, diseases; Nephritis
Nerves: See also under Neuritis; Paralysis
Cell: See Neurons
Optic: See also Neuritis, optic
optic, alterations of, and oxycephaly, 402
optic, aplasia of, 893
roots; polyradiculoneuritis with dissociation in protein content and cellular content of spinal fluid, 883
vagus; certain therapeutic effects of whole suprarenal gland by mouth, 888
Nervous and Mental Disabilities: See also Chorea; Encephalitis; Feeblemindedness; Idiocy; etc.
enuresis and other factors in normal and in psychotic persons; comparative study of incidence and intercorrelations, 428
psychiatry and pediatrics, 426
school life and after for handicapped child, 1099
Nervous System: See also Brain; Cerebellum; Nerves; Reflex; Spinal Cord; etc.
Diseases: See Epilepsy; Mental Diseases; etc.
effect of climate on convalescent children, 1136
emotional and nervous diseases in pediatrics from clinical and social points of view, 1339
erythroblastic (Cooley's) anemia and neurologic complications (status dysmyelinatus), *362
insensible perspiration in children; influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71
newer aspects of nervous disorders in avitaminosis, 852
rapid death from eczema in infancy; role of neurovegetative system, 439
therapy of encephalitis and related neurotropic infections of nervous system, 1120
vegetative regulation of red blood cells in anemia, 1112
Neuralgia, trigeminal, new method in treatment, 887
Neuritis caused by prophylactic vaccination against tetanus, 194
Neuroblastoma of left adrenal with intrathoracic metastases, 432
Neurofibromatosis, congenital ectodermoses in infancy and childhood, 1346
congenital nevi and dermatolysis (atypical Recklinghausen's disease), 439
four syndromes of "phakomatoses," 647
inheritance of neurofibroma (Recklinghausen), 650
Neurons, depth and nerve-cell content of supragranular cortex in normal and mentally defective persons, 426
embryonic and postembryonic development of cortex, 596

Neurosyphilis: See Tabes Dorsalis
Neutropenia: See Granulocytopenia
Neutrophils: See Leukocytes
Nevi, congenital, and dermatolysis (atypical Recklinghausen's disease), 439
Newborn Infants, *1. See also under names of diseases, as Anemia; Asphyxia; Conjunctivitis; Cyanosis; Erysipelas; Gangrene; Impetigo; Jaundice; Meningitis; Pneumothorax; Syphilis; Tetany; etc.
anemia of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
biochemical studies of blood and spinal fluid in, 1103
biometric data on newborn of province of Perugia, 859
bronchopulmonary staphylococcic infection of newborn; serotherapy, then vaccine therapy; cure, 608
capsular hemorrhages of adrenals in, 186
clinical and histologic investigations of so-called witch's milk lactation with attention to its relation to birth weight of infant, 1312
complement in newborn children, in infants and in fetuses, 860
complemental feeding and care of skin in, 607
congenital anomaly of valve of Bauhin and of appendix as cause of peritonitis in, 1312
congenital ichthyosis, 1312
cyanosis in, 403
effect of various feedings on loss and gain of weight during newborn period, 912
epidemic diarrhea of, 662
gastrointestinal hemorrhage of newborn; two cases, 1311
importance of heart block in, 403
jejunal intussusception of, *558
meningeal hemorrhages of, 859
perforation of gastrointestinal tract of, *371
pneumococcic meningitis of, 403
precaution in handling of, 1312
rupture of stomach in, 185
sedimentation time and other blood factors in, 185
traumatic separation of upper epiphysis of femur; typical birth injury, 609
urea content of brain of, 1311
varicella in, *564
Nipples: See Breast
Nitrogen, assimilation of protein by young children with nephrotic syndrome; effect of dietary fat and carbohydrate on nitrogen balance, *935
assimilation of protein by young children with nephrotic syndrome; effect of nephrotic crises on assimilation of nitrogen, *939
atmosphere of reduced nitrogen with respiratory obstruction, 1351
continuous calcium and nitrogen balances before and after menarche, 672
effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on calcium retention, *1186
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on nitrogen retention, *965
influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
long-time study of nitrogen, calcium and phosphorus metabolism on low protein diet, 1093
long-time study of nitrogen, calcium and phosphorus metabolism on medium protein diet, 1093
Nitrogen—Continued
metabolism and utilization of foodstuffs in children receiving diets containing vegetable protein, 851
residual, total serum protein and albumin-globulin quotient in dyspeptic toxicosis of infants, 1334
Nodes: See Lymph Nodes; Rheumatic Fever; etc.
Noma: See Stomatitis, gangrenous
Nose: See also Nasopharynx; Rhinitis; Smell; etc.
Diphtheria: See Diphtheria
ephedrine in physiologic vehicle and lateral head low posture in treatment of nose and sinuses, 1328
nasal allergy, 220
septum surgery in children, 1113
Nungester, W. J.: Distribution of pneumococci in experimentally infected rats and penetration of antiserum into lesions, 1355
Nurses and Nursing, disabling diseases of childhood; their characteristics and medical care as observed in 500,000 children in 83 cities canvassed in National Health Survey of 1935-1936, *1157
scarlet fever immunizations in group of nurses, 611
Nutrition: See also Diet and Dietetics; Dystrophy; Food; Infant Feeding; Metabolism; Vitamins; etc.
as affected by economic level, 1098
beriberi and other symptoms of nutritional disturbance in infant, 182
considerations in infant nutrition, 1304
mode of action of vegetables in feeding of infants, 395
muscular hypertonicity in infants of nutritional cause, 179
newer developments in child nutrition, 850
nutritional factors in dental development, 222
nutritional problems of childhood, 597
nutritional requirements in inanition, 669
of preschool child, 604
physical indices and clinical assessments of nutrition of school children, 398
predisposition to diarrhea, 210
treatment of nutritional disorders in infants with ascorbic acid, 854
utilization of carbohydrate in human undernutrition, 1094
wasting or marasmus, 1303
Nystagmus, congenital heredofamilial disease, consisting of rhythmic tremor of head, eyes and upper extremities, 631

1162 F: See Gonorrhea
Obesity, 849. See also Laurence-Biedl Syndrome; Pituitary Body, diseases
in childhood; basal metabolism and serum cholesterol of obese children, *1001
in childhood; physical growth and development of obese children, *457
infantile, treatment of, 180
pseudohermaphroditism, adiposity, polyuria and hyperglycemia; infundibulotuberian syndrome, 630

OBITUARIES:
Baldwin, John C., 595
Hamilton, Howard B., 1090
Park, William Hallock, 175
Tumpeer, Isidor H., 1371
Wollstein, Martha, 1301

Obstetric Injuries: See under Newborn Infants
Obstetrics: See also Labor; Pregnancy
panel discussion on cyanosis of newborn; obstetric aspects of cyanosis, 1311
thymus and thymicolymphatic constitution; clinical aspects as seen by obstetrician, 1358

Oil: See also Cod Liver Oil; Fat; etc.
histologic study of hypervitaminosis D; relative toxicity of vitamin D of irradiated ergosterol and tuna liver oil, 1095
Olney, M. B.: Comparative studies on use of standardized protamine zinc and crystalline insulin, 919
Oppenheim's Disease: See Myatonia
Optic Chiasm, arachnoiditis of optic chiasm cured by encephalographic examination, 1336
Oranges, studies on Hunan local food products; chemical composition and vitamin C content of Hunan citrus fruits, 604
Orbit, myelogenous chloroma, 1328
Organs, correlation of fetal body length with weights of organs, 1102
Orthodontia: See under Teeth
Orthopedics: See under Osteomyelitis; etc.
Orthoptics: See Strabismus
Orthostatism: See Posture
Osgood, E. E.: Total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
Total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Osgood, M. W.: Total, differential and absolute leukocyte counts and sedimentation rates of healthy children 4 to 7 years of age, *61
Total, differential and absolute leukocyte counts and sedimentation rates for healthy children; standards for children 8 to 14 years of age, *282
Osler's Disease: See Telangiectasis
Ossification: See also Bones, growth; etc.
rate of appearance of ossification centers from birth to age of 5 years, *949
Osteitis fibrosa, generalized, management of, 645
Tuberculous: See Bones, tuberculosis
Osteochondritis deformans juvenilis; pathogenesis and nature of Perthes' disease, 1130
deformans juvenilis; some remarks on Perthes' disease, especially with regard to treatment, 645
dissecans of hip, 217
juvenile femoral osteochondritis with endocrine difficulties, 1129
Osteodystrophia Fibrosa: See Osteitis fibrosa
Osteogenesis Imperfecta: See Bones, fragility
Osteomyelitis, acute, outlawing of emergency operations in treatment, 435
acute, source of staphylococcal infection in, 433
acute, treatment, 435
acute, treatment in adolescents, 1344
petrosquamous, in infants, 1329
relapses of staphylococcic origin, 891
scorbutic subperiosteal hemorrhage complicated by osteomyelitis and pyarthrosis, 1095
transfusions in pediatric orthopedics, 1134
treatment during period of growth, 891
Osteoporosis: See Bones, atrophy
Osteopsathyrosis: See Bones, fragility
Osteosynthesis: See Spine
Osterlin, M. L.: Gross intestinal hemorrhage due to pathologic condition in Meckel's diverticulum in 2 infants under 1 year of age, 1354
Otitis Media: See also Mastoiditis
acute, 202
bacteriologic and clinical studies of complications of surgically handled acute otitis media, 875
from pediatrician's viewpoint, 418
intracranial complications of ear disease, 624
otitic meningitis due to Streptococcus hemolyticus; operation and recovery, 630
Otitis Media—Continued
para-aminobenzenesulfonamide and its derivatives in treatment of beta hemolytic streptococcus infections of middle ear and mastoid; report of 6 cases in children, 202
pediatric treatment of otologic sepsis, including meningitis, 1113
problems of suppurative petrositis and its surgical treatment together with outline of prophylaxis and treatment of otitic meningitis, 203
sedimentation reaction in otitis media and its complications, 419
symposium; what is justifiable to do in otitic meningitis; brief (historical) review of operative treatment of purulent meningitis; surgical indications in suppurative meningitis; surgical treatment of meningitis; sulfanilamide and related compounds in treatment of meningitis secondary to ear and sinus infections, 623
tuberculous, 625
Otorhinolaryngology, otolaryngologic suggestions in pediatric practice, 202
thymus and thymicolymphatic constitution; clinical aspects as seen by otolaryngologist, 1359
Ovary: See also Estrogens; Gonads; Hormones
effect of irradiation on function in young girls, 1123
hormones; influence of lecithin and of lutein on hypoglycemic action of insulin, 642
progesterone is androgenic, 1127
Oxycephaly: See Acrocephaly
Oxydase and Peroxydase, hemosedimentation of healthy lactating women (Arakawa reaction), 1328
Oxygen: See also Respiration
in Blood: See Blood, oxygen
lack; its causes, effects and relief, 225
relative value of pure oxygen and of carbon dioxide mixtures in experimental resuscitation, 858
uptake of tissues in vitamin deficiencies, 1095
Oxyuriasis, appendicitis from Oxyuris infestation, 881

Pachman, D. J.: Enterogenous intramural cysts of intestines, *485
Pain: See Abdomen, pain; etc.
Palate, hyperpyrexia following operation for cleft lip or palate, *320
tuberculosis of hard palate, 197
Palsy: See Paralysis
Pancreas, hematoneurologic syndrome with disturbance of dextrose metabolism from thymicopancreatic dysfunction, 642
hyperinsulinism, 1343
hypoglycemia in 2 year old child with subtotal removal of pancreas, 1367
peptic ulcer perforating into pancreas, 880
pituitary therapy and carbohydrate tolerance, 1369
Papilledema: See Neuritis, optic
Paralysis: See also Extremities, paralysis; Poliomyelitis; etc.
agitans, late forms of amaurotic idiocy showing picture of, 1122
causation in diphtheria, 406
cerebral; hypoplasia of cerebral motor nuclei or nerves, combined with clinocephaly, 1120
congenital, of facial nerve and abducens nerve, 1336
experimental postdiphtheritic paralysis and vitamin B_1, 1107
facial, epidemic due to poliomyelitis, 1106
General: See Dementia Paralytica
Infantile: See Poliomyelitis
progressive facial palsy produced by intratemporal epidermoids, 900
Respiratory: See Respiration, disturbances

Paralysis—Continued
spastic conditions in children, 630
Werdnig-Hoffmann: See Atrophy, muscular
Paraplegia: See Extremities, paralysis; Spine, tuberculosis
Parasites: See Intestines, parasites; and under names of parasitic diseases, as Ascariasis; Oxyuriasis; etc.
Parathyroid, comparison of effects of A. T. 10 (dihydrotachysterol) and vitamin D on calcium and phosphorus metabolism in hypoparathyroidism, 600
effect of vitamin D on calcium and phosphorus metabolism; studies on 4 patients, 600
effect on growth rate of thyroparathyroidectomy in newborn rats and of subsequent administration of thyroid, parathyroid and anterior hypophysis, 640
hyperparathyroidism in children; report of 2 cases, *540
Parathyroidectomy, Results: See Tetany
Paratyphoid, epidemic of alimentary intoxication due to organism of paratyphoid B group, 1333
Paresis: See Dementia Paralytica
Parotitis, eosinophilic bodies in blood of mumps, 861
prophylactic and therapeutic value of placental extract in regard to measles, scarlatina and mumps, 190
studies on period of incubation of mumps, 863
Parsons, C. G.: Circulatory function in anemias of children; roentgenographic measurement of cardiac size, *250
Parturition: See Labor
Pediatric societies, directory of, 230, 450, 684, 924, 1150, 1374
Pediatrics: See also Children; Hospitals; Infants; and under names of specific diseases and psychiatry, 426
inhalation therapy and ultraviolet rays in preventive pediatrics, 1349
otolaryngologic suggestions in, 202
pediatrist and surgeon, 225
Pelger's Nuclear Anomaly: See Leukocytes, count
Pelizaeus-Merzbacher's Disease: See Brain, diseases
Pellagra: See also Vitamins, B
recent observations on treatment of 600 pellagrins with emphasis on use of nicotinic acid in prophylaxis, 396
treatment with reference to use of nicotinic acid, 396
Pelvis, treatment of fracture in childhood, 224
Pemphigus vulgaris in children, 1133
Peptic Ulcer, chronic, in children, 1332
navel colic, 629
pathologic reports from Children's Hospital, Melbourne, 628
perforating into pancreas, 880
perforation of gastrointestinal tract of newborn infant, *371
probable duodenal ulcer in child, 628
rupture of stomach in newborn, 185
Pericarditis and rheumatic endocarditis, 871
Rheumatic: See under Rheumatic Fever
Periostitis, vitamin C deficiency and periostitis of both ulnae? scurvy, 1097
Peritonitis, congenital anomaly of valve of Bauhin and of appendix as cause of peritonitis in newly born, 1312
due to ruptured acute appendicitis in children; influence of delay on operative mortality, 1332
experimental investigation on relationship of vitamins to infection, 602
fetal, 857
gonococcal, 410
prontosil and treatment of spreading peritonitis in dogs, 208
streptococcal, with septicemia, 880
Peritonsillitis: See under Abscess
Personality, ocular and dental malformations and disturbances of personality in 2 brothers with hereditary syphilis, 869
Perspiration, Insensible: See Metabolism
Perthes' Disease: See Osteochondritis deformans juvenilis
Petrositis: See Temporal Bone
Petrous Bone: See under Temporal Bone
Pertussis: See Whooping Cough
Phagocytes and Phagocytosis, phagocytosis of tubercle bacilli in children (Otani's method), 620
Phakomatoses: See Neurofibromatosis
Phalanges: See Fingers and Toes
Pharynx: See Nasopharynx
Phenol; phenol-contaminated waters and their physiological action, 177
Phonocardiography: See under Fetus
Phosphatase in Blood: See under Blood
Phosphate, Therapy: See Rickets
Phosphorus and Phosphorus Compounds, chemical composition of transverse metaphysial bands produced in growing bones by phosphorus ingestion, 1343
comparison of effects of A. T. 10 (dihydrotachysterol) and vitamin D on calcium and phosphorus metabolism in hypoparathyroidism, 600
effect of vitamin D on calcium and phosphorus metabolism; studies on 4 patients, 600
in Blood: See under Blood
long-time study of nitrogen, calcium and phosphorus metabolism on low protein diet, 1093
long-time study of nitrogen, calcium and phosphorus metabolism on medium protein diet, 1093
phosphorus esters in human, cow's and goat's milk, 180
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Photosensitization: See Ultraviolet Rays
Pigmentation, cutaneous; tanning from ultraviolet irradiation brought out by blood transfusion, *1047
Pilocarpine, insensible perspiration in children; influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71
Pineal Gland, experimental phases of pineal problem, 1127
tumor, 432
Pink Disease: See Acrodynia
Pinworm Infestation: See Oxyuriasis
Pituitary Body: See also Hypothalamus
Diseases: See also Acromegaly; Laurence-Biedl Syndrome; Obesity; etc.
diseases; histologic examination of hypothalamus in ordinary obesity, in Morgagni's and Gunther's syndromes and in morbus Cushing, 1120
diseases; juvenile adiposogenital dystrophy, neurologic and psychopathologic aspects, results of organotherapy and psychotherapy, 889
effect on growth rate of thyroparathyroidectomy in newborn rats and of subsequent administration of thyroid, parathyroid and anterior hypophysis, 640
Fröhlich syndrome; report of original case, *1282
function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
"neuro-uveo-parotitis," 632

Pituitary Body—Continued
pituitary dwarfism treated with antuitrin-G, 430
thymus studies of some changes in gonads and pituitary following its destruction by roentgen irradiation, 890
urinary thyrotropic hormone, 640
Pituitary Preparations, lactogenic hormone, 1126
pituitary therapy and carbohydrate tolerance, 1369
role of iodine (potassium iodide) in experimental hyperthyreoidism, 642
treatment of nurslings' atrophy with extracts of liver and hypophysis, 851
Placenta, experimental studies on congenital allergy, 898
placental globulin in modification of measles, 187
placental interchange; comparison of total base concentration of fetal and maternal blood at parturition, 858
placental transfer of sulfanilamide, 184
prophylactic and therapeutic value of placental extract in regard to measles, scarlatina and mumps, 190
prophylaxis against measles, 190
some facts about congenital malaria, 1108
use of placental extract in epidemic of measles, 407
Plastic Surgery: See Surgery, plastic
Play-interview; method of studying children's attitudes, *1199
Pleura, manifestations of perforation of esophagus, *1261
Pleurisy, action of intrapleural injections of hypertonic solutions of sugar in pleuritis exsudativa and in empyema, 421
as allergic manifestation of primary tuberculosis, 414
Plumbism: See Lead, poisoning
Pneumococci, demonstration in throats of normal infants and of infants with pneumonia or infection of upper respiratory tract, 1330
distribution in experimentally infected rats and penetration of antiserum into lesions, 1355
Infections: See also under Meningitis; Peritonitis; Pneumonia
meningitis caused by pneumococcus type III: observations on sulfanilamide therapy, *274
new method of obtaining sputum from infants and children thought to have pneumococci in nasopharynx, 1139
pneumococcic meningitis of newborn, 403
precise evaluation of therapeutic antipneumococcal sera, 847
Pneumography: See Brain, roentgenography
Pneumonectomy: See Bronchiectasis
Pneumonia: See also Bronchopneumonia
acute lung conditions with reference to their treatment in infants, 1330
clinical and experimental studies of lipoid pneumonia, 654
comparison of etiology, death rates and bacteremic incidence in more frequent primary pneumonias of infants, children and adults, 204
complicated by acute hemorrhage and ulcerative gastroenteritis in child, *1265
demonstration of pneumococci in throats of normal infants and of infants with pneumonia or infection of upper respiratory tract, 1330
diphtheritic or staphylococcic croup and simultaneous pneumococcic pneumonia, *817
distribution of pneumococci in experimentally infected rats and penetration of antiserum into lesions, 1355
experimental study of treatment of pneumococcal toxemia with special carbon preparation, 865
in infancy and childhood, 877
Pneumonia—Continued
incidence of types and therapeutic results in cases in infancy and childhood in Detroit, 658
intramuscular administration of antipneumococcal serum in infants and children, 1330
lobar, serum therapy in, 1115
lobar, treatment with M. & B. 693, 206
localized bullous emphysema associated with pneumonia in children, 1139
nature of allergic and nonallergic lobar pneumonias, 208
primary bronchogenic staphylococcic pneumonia, 205
rheumatic lung, 420
serum treatment of pneumonia due to pneumococcus type XIV, 656
Tuberculosis: See Tuberculosis, pulmonary
Pneumothorax: See also Empyema; Tuberculosis, pulmonary
basal metabolism of tuberculous children; children with pneumothorax, *957
Poisons and Poisoning: See also under names of poisonous substances, as Lead; Lye; Mercury; Strychnine; etc.
poisoning in childhood, 1136
Poliomyelitis: See also Encephalomyelitis
acute anterior poliomyelitis following tonsillectomy and adenoidectomy with reference to bulbar form, 1105
anterior, in China, 408
cortical form of Heine-Medin's disease, 1315
dehydrating therapy in cases of infantile paralysis, 1317
detection of virus in nose and throat and gastrointestinal tract of humans and monkeys, 610
early lesions after intranasal inoculation with comments on their relationship to early clinical manifestations and to nonparalytic cases, 188
early treatment, 862
effect of supersonic waves on virus, 409
epidemic of facial paralysis due to, 1106
epidemiological study in District of Columbia, 611
experimental, case of relapse in, 1314
experimental, second attacks in Macacus rhesus monkeys; immunity or lack of immunity to Philadelphia 1932 strain of virus, *41
extension of disease process in Heine-Medin's disease, 1316
future of chemoprophylaxis as measure for practical control, 1104
method for concentration of virus in nasopharyngeal washings, 609
nasal spraying with zinc sulfate solution in prophylaxis; diphtheria immunization as partial preventive, 190
persistent anosmia following zinc sulfate nasal spraying, 201
practitioner's viewpoint, 189
vitamin C nutrition and susceptibility to diphtheria as possible factors in epidemiology of, 1314
Pollen: See Anaphylaxis and Allergy: Asthma; Hay Fever
Polycythemia, histologic examination of hypothalamus in ordinary obesity, in Morgagni's and Günther's syndromes and in morbus Cushing, 1120
primary, with leukemic manifestations, *146
Polyradiculoneuritis: See Nerves, roots
Polyserositis: See Serositis
Polyuria: See Urination, excessive
Pons Varolii, differential diagnosis of pontile tumors and encephalitis, 1336
gliomas; clinical and pathologic characteristics, 1335
Porphyria: See Hematoporphyria

Posture, ephedrine in physiologic vehicle and lateral head low posture in treatment of nose and sinuses, 1328
orthostatic hypotension; report of 2 cases in which patients were children, 199
some common postural deformities and their treatment by exercises and manipulation, 1134
Potassium Iodide: See Iodine and Iodine Compounds
Pott's Disease: See under Spine
Precocity: See Adolescence
Pregnancy: See also Fetus; Labor; Maternity; Obstetrics
attempt to increase resistance to pertussis in newborn infants by immunizing their mothers during pregnancy, 1104
blackwater fever following administration of "atebrin" in pregnant woman, 1318
eclampsia-like syndrome occurring in pregnant dogs and rabbits following renal artery constriction, 1339
Multiple: See also Twins
multiple pregnancies in woman with congenital cyanosis due to heart disease, 1326
urine in; hormone treatment of imperfect descent of testis; further observations, 429
urine in; human serum response to gonadotropic hormone (pregnancy urine extract), 1127
Premature Infants, biometric study of growth in, 1101
causes of prematurity; review of literature, *586
constitutional factors in etiology of prematurity, 1101
development of, 1101
dislocation of ocular lenses of premature infant; dolichostenomelia? 646
early general and feeding care, 856
eosinophilia in, 1327
experiments with wheat germ oil (vitamin E) in nutrition of, 1102
factors influencing mortality rate of, 1307
treatment of grave condition in premature infant with estrogen, 402
Presentation: See under Labor
Progesterone: See Ovary
Prontosil: See Peritonitis; Sulfanilamide and Sulfanilamide Derivatives
Proseptasine: See Meningitis; Sulfanilamide and Sulfanilamide Derivatives
Prostigmine, Therapy: See under names of diseases, as Myasthenia
Protamine Insulin: See under Diabetes Mellitus; Insulin
Protein: See also Nitrogen; and under names of proteins, as Albumin; etc.
assimilation by young children with nephrotic syndrome; effect of dietary fat and carbohydrate on nitrogen balance, *935
assimilation by young children with nephrotic syndrome; effect of nephrotic crises on assimilation of nitrogen, *939
in Blood: See Blood, proteins
in Cerebrospinal Fluid: See under Cerebrospinal Fluid
in Urine: See Albuminuria
long-time study of nitrogen, calcium and phosphorus metabolism on low protein diet, 1093
long-time study of nitrogen, calcium and phosphorus metabolism on medium protein diet, 1093
metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
sulfur and protein metabolism of naturally and artificially fed infants, 1305
Prothrombin: See Blood coagulation
Protozoa: See Intestines, parasites
Pseudohermaphroditism: See Hermaphroditism
Pseudoleukemia, infantile; erythremic myeloid osteopathy (erythroblastic anemia), 219
infantile; erythremic myelosis, 874
infantile; erythroblastic (Cooley's) anemia and neurologic complications (status dysmyelinatus), *362
infantile; hemolytic anemias of childhood, 621
pseudoleukemic splenic anemia in syphilitic infant, 869
Psoriasis, treatment of, 898
Psychiatry and pediatrics, 426
Psychoanalysis, adaptation to reality in early infancy, 636
play-interview; method of studying children's attitudes, *1199
Psychology: See also Mental Tests; Personality baby health and child welfare, 213
Psychotherapy, juvenile adiposogenital dystrophy, neurologic and psychopathologic aspects, results of organotherapy and psychotherapy, 889
Pubertas Praecox: See under Adolescence
Puberty: See Adolescence
Public Health: See Child Welfare; etc.
Pulmonary Valve, tuberculous endocarditis of, 871
Pulsator: See Respiration, artificial
Pulse, Bigeminal: See Arrythmia
Purpura, allergic, 221
glomerulotubular nephropathy in Schoenlein-Henoch purpura, 416
Haemorrhagica: See also Blood, platelets; Diathesis, hemorrhagic
haemorrhagica; further studies on Werlhof's morbus maculosus; hitherto unknown fat-soluble T factor which caused thrombocytosis, 873
haemorrhagica; thrombocytopenic purpura; 3 case reports, 199
Thrombocytopenic; Thrombopenic: See Purpura, haemorrhagica
Pyarthrosis: See Joints, diseases
Pyelography: See Kidneys
Pyknolepsy, diagnostic differentiation of epilepsy from pyknolepsy and salaam convulsions in children, 884
Pylorus, congenital hypertrophic stenosis, 422
congenital stenosis and results of treatment, 629
congenital stenosis in first and second cousins, 209
congenital stenosis with cardiostenosis in child aged 4 days, 1117
medical or surgical therapy of congenital pyloric hypertrophy (pylorospasm)? 1333
roentgen diagnosis of hypertrophic pyloric stenosis in infants, 1334
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Pyorrhea, diet and its relation to dental disease, 1134

"Q" Fever: See Rickettsia
Quinine, does animal experimentation show similar changes in ear of mother and fetus after ingestion of quinine by mother? 605
rectal absorption of, 226
Therapy: See Myotonia; Stomatitis, gangrenous

Rabies, diagnosis of, 866
epidemiologic and immunologic experiments, 614
experiments on antirabic immunization, 866
Races, body measurements of Texas school children, 400

Rachitis: See Rickets
Radiations: See Ultraviolet Rays; and under names of various diseases
Radiculitis: See Nerves, roots
Radius, congenital bilateral radio-ulnar synostosis, *1259
Ragweed: See Hay Fever
Ramond-Still-Chauffard Disease: See Arthritis deformans
Rapoport, S.: Role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
Rash: See Roseola
Rat Bite Fever in children, 1321
recognition in children with reference to hematologic and immunobiologic data, 194
Ratner, B.: Experimental asthma, *699
Reading disability, 1121
Recklinghausen's Disease: See Neurofibromatosis; Osteitis fibrosa
Rectum: See also Enemata
absorption of quinine, 226
absorption of vitamin C, 397
rectosigmoidoscopic examinations in children's clinics, 628
Reflex, Adie's syndrome in child, 883
experimental studies on conditioned salivary reflexes in children, 214
protective, 633
Respiration: See also Dyspnea
artificial; relative value of pure oxygen and of carbon dioxide mixtures in experimental resuscitation, 858
atmosphere of reduced nitrogen with respiratory obstruction, 1351
coordination of sucking center and respiratory center in human suckling, 596
disturbances; respiratory paralysis due to diphtheria successfully treated by means of respirator, 407
evidence of fetal respiration in utero, 1308
Physiology: See Vital Capacity
respiratory changes, effect on blood pressure, *114
Respiratory Quotient: See Metabolism
Respiratory Tract: See also Bronchi; Nasopharynx; Nose; etc.
clinical, anatomopathologic and bacteriologic study of 2 cases of acute stenosing laryngotracheobronchitis, Chevalier Jackson type, 626
demonstration of pneumococci in throats of normal infants and of infants with pneumonia or infection of upper respiratory tract, 1330
Diseases: See also Pneumonia; etc.
diseases; cold vaccines; evaluation based on controlled study, 201
occurrence of blood in air passages after tonsillectomy, 875
source of staphylococcal infection in acute osteomyelitis, 433
vitamin C and acute infections of upper respiratory tract, 1329
Resuscitation: See Respiration, artificial
Reticulocytes: See Erythrocytes
Reticuloendothelial System: See Anemia, splenic; Jaundice; Liver; Mononucleosis, infectious; Spleen; etc.
Retina, familial glioma, 436
hemorrhage of brain and retina in scurvy, 600
hemorrhages in association with meningeal hemorrhage of newborn, 1103
hitherto unknown hereditary ocular disease (degeneratio hyaloidea-retinalis hereditaria) observed in canton of Zurich; report of case, 1131
Retinitis, nephritis with albuminuric retinitis and patent ductus arteriosus, 638
Pigmentosa: See also Laurence-Biedl Syndrome
pigmentosa; melanosis retinae, 894
pigmentosa; pigmentary degeneration of retina as dominant hereditary characteristic, 646
Rhabdomyosarcoma: See under Sarcoma
Rheumatic Fever, acute, clinical observations in, 1322
acute, metabolism of lactic acid in, 1138
aminopyrine treatment of rheumatic endocarditis associated with active rheumatic infection, 411
congenital and acquired heart disease and its retrospective diagnosis, 200
cytologic studies on, 191
effect of sulfanilamide on rheumatic fever and chorea, 615
etiology of chorea; its relation to rheumatic fever and heart disease, 424
hemolytic streptococcus in etiology of rheumatic fever and rheumatoid arthritis, 192
in vitro experiments on effect of added blood serum and blood plasma on sedimentation rate, 1137
incidence of rheumatic infections in children of Oregon, 410
left upper abdominal pain on effort in juvenile rheumatic disease; new symptom of low grade active phase, 865
newer concepts in diagnosis and treatment of juvenile rheumatism, 865
rheumatic endocarditis and pericarditis, 871
rheumatic infection, 615
rheumatic infection in children of Uruguay, 617
rheumatic infections in childhood; effect of surgical operations on blood sedimentation rate, 410
rheumatic lung, 420
rheumatic subcutaneous nodule and simulating lesions, 1317
seasonal and regional factors in acute rheumatic fever and rheumatic heart disease, 192
Rheumatism: See also Arthritis
irritation and toxic susceptibility in course of rheumatic infection, 412
rheumatic problem in Great Britain, 616
sensitivity to tuberculin in rheumatic disorders, 412
treatment in childhood, 1320
Rhinitis: See also Respiratory Tract, diseases acute, and acute sinusitis, 202
Vasomotor: See also Hay Fever
vasomotor; nasal allergy, 220
Ribs, abnormalities; unusual roentgenologic findings in infants, 226
Rickets: See also Achondroplasia; Vitamins, D
and blood transfusion, 603
can rickets be eliminated from large cities? 1305
Celiac: See Celiac Disease
changes of temporal bone in rickets and in "renal rickets," 1098
clinical picture in practice, 183
experimental, 599
further investigations in single dose vitamin therapy of, 1097
in Egypt, 602
infantile, phosphate solutions in treatment, 668
is rickets due simply to vitamin deficiency? 1098
late rickets with glycosuria and organic acid acidosis (Fanconi type), 673
phosphorus and glycerophosphatase of blood of rachitic children; variations observed in course of treatment with ultraviolet radiation, 1097
problem of curing rickets, 1307

Rickets—Continued
Renal: See Dwarfism
role of acid-soluble phosphorus compounds in red blood cells in experimental rickets, renal insufficiency, pyloric obstruction, gastroenteritis, ammonium chloride acidosis and diabetic acidosis, *1072
shock treatment with vitamin D_2 and D_3, 603
significance of rachitic bone changes in early childhood for later life, 1307
summer and winter rickets in Hamburg, 183
syndrome consisting of affections of kidney, stunted growth, rickets and disturbed cystine metabolism, 215
therapy and prophylaxis with single large dose of vitamin D_2 and D_3, 1307
treatment with vitamin D_2, 1097
ultraviolet ray of diffuse daylight and its therapeutic utilization, 397
use of vitamin D preparations in prevention and treatment of disease, 181
Rickettsia: See also Rocky Mountain Spotted Fever
laboratory infections with rickettsia of "Q" fever, 1318
"Q" fever; factors affecting appearance of rickettsiae in mice, 1318
Ritter's Disease: See Dermatitis exfoliativa
Rivanol: See Eczema
Robertson, E. C.: Air contamination and air sterilization, *1023
Robinow, M.: Accuracy of clinical determinations of blood pressure in children with values under normal and abnormal conditions, *102
Rocky Mountain Spotted Fever on Long Island, 865
Roentgen Rays, Therapy: See under names of various diseases, as Sarcoma; etc.
Roseola, exanthema subitum (roseola infantum), *983
Ross, J. R.: Treatment of chronic eczema in children, 1145
Ross, S. G.: Vitamin D intoxication in infancy, 666, 1142
Roundworms: See Ascariasis
Rubella, German measles as virus disease, 409
Rubeola: See Measles; Rubella
Rubin, H. S.: Basal metabolism of tuberculous children; children with pneumothorax, *957
Rubinstein, R. I.: Congenital leukemia with "chloroma," *332

Sachs-Tay's Disease: See Idiocy, amaurotic
St. Vitus' Dance: See Chorea
Salicyl Compounds, gastroscopic observation of effect of aspirin and certain other substances on stomach, 209
Salmonella enteritis in infants, 423
Panama: See Food, poisoning
suipestifer; septic illness of young infant caused by Salmonella suipestifer bacilli, 1320
Salts, Iodized: See Iodine and Iodine Compounds
Samwick, A. A.: Congenital leukemia with "chloroma," *332
Sanes, S.: Anomalous drainage of pulmonary veins into coronary sinus, *354
Sanford, H. N.: Effect of various feedings on loss and gain of weight during newborn period, 912
Sarcoid, Boeck's, 218
osteitis tuberculosa multiplex cystoides and sarcoid lesions, 643
Sarcoma: See also Cancer; Tumors; and under names of organs and regions
effect of roentgen irradiation on sarcoma of cerebellum, 1336
of intestine in children, 879
renal adenosarcoma and subcutaneous elimination pyelography, 640
rhabdomyosarcoma of urinary bladder, 1339
Scalp, congenital nevi and dermatolysis (atypical Recklinghausen's disease), 439
diseases; some experiences with mercury vapor lamp, 1132
enormous hematoma of scalp resulting in death, 1311
Scapula, brevicollis, 184
Hand-Schüller-Christian disease of, 1325
Scarlatina: See Scarlet Fever
Scarlet Fever: See also Streptococci
anaphylactic reactions during Dick test and active immunization with scarlet fever toxoid, 191
appearance of reticulocytes in, 409
cerebral complications in, 863
Dick reaction and active immunization with toxoid among school children, 190
disturbance of gait, probably following scarlatinal encephalitis, 1314
extrasystolic arrhythmia in, 1317
heart block in, 1317
immunization, 611
immunization and treatment, 611
immunizations in group of nurses, 611
immunologic properties of scarlatina convalescent serum, *1039; correction, 1372
in Chile, 863
intradermic immunization against, 861
local and peripheral leukocyte counts in certain infectious diseases, 1315
observations on patients during last 5 years, 864
prophylactic and therapeutic value of placental extract in regard to measles, scarlatina and mumps, 190
prophylaxis in institutions, 1316
scarlet fever heart, 1316
studies in immunity; spontaneous changes in Dick and Schick reactions of children, *734
sulfanilamide and serum in treatment and prophylaxis of, 187
survey, 1313
treatment of diphtheria and scarlet fever by active immunization, 406
Scars: See Cicatrix
Schaumann's Disease: See Lymphogranuloma
Schick Test: See Diphtheria
Schizophrenia: See Dementia Praecox
Schlutz, F. W.: Physical fitness in child as reflected by changes in cardiovascular system and in blood serum during exercise, 676
Schnute, L.: Sequelae of postinfectional encephalitis with reference to mental involvement, 1352
Schoenlein-Henoch's Purpura: See Purpura
Schools, ascorbic acid metabolism of very small children and those of school age, 602
body measurements of Texas school children, 400
conduct of school epidemic of measles, 407
Dick reaction and active immunization against scarlet fever with toxoid among school children, 190
external diseases of eye in school children, 645
incidence of tonsillectomy in school children, 203
physical indices and clinical assessments of nutrition of school children, 398
recommendations issued by fourteenth Congress of Hygiene, 399
school health program and physician, 401
school life and after for handicapped child, 1099
tuberculosis in American schools and colleges, 868
verification of tests of hypovitaminosis C in children of school age, 1306
Schroeder, P. L.: Behavior problems of adolescents, *168

Schüller-Christian Syndrome, 1111
Hand-Schüller-Christian disease of scapula, 1325
Sclera, syndrome of blue sclerotics (van der Hoeve), 894
Scleredema: See Scleroderma
Scleroderma, case of scleredema, 439
subcutaneous fat necrosis of newborn, 404
Sclerosis, multiple, in children, 884
tuberous; congenital morphologic abnormalities of brain in case of abortive tuberous sclerosis; functional implications and bearing on pathogenesis of so-called genuine epilepsy, 1119
Scoliosis: See Spine, curvature
Scorbutus: See Scurvy
Scrofula: See Tuberculosis
Scurvy: See also Vitamins, C
and vitamin C, 853
Barlow's disease in Uruguay, 602
comparison of methods for detecting and grading subclinical scurvy, 181
experimental, of guinea pig, 603
hemorrhage of brain and retina in, 600
lactic acid formation by muscles of scorbutic guinea pigs, 1343
Moeller-Barlow disease, 853
scorbutic subperiosteal hemorrhage complicated by osteomyelitis and pyarthrosis, 1095
Seasons, does birth of mentally defective children show seasonal fluctuation? 1339
seasonal and regional factors in acute rheumatic fever and rheumatic heart disease, 192
study of breed and seasonal variations in ascorbic acid content of certified milk from Guernseys and Holsteins, 1096
summer and winter rickets in Hamburg, 183
winter diarrhea in infants, 879
Seborrhea, seborrheic dermatitis, 648
Seckel, H. P. G.: Prevention of diphtheria, *512
Secretions, Internal: See Endocrine Glands
Section on Pediatrics of American Medical Association, meeting of, 1371
Segal, H. N.: Cardiac irregularities in infants, 1147
Sensitization: See under Anaphylaxis and Allergy; Immunity; etc.
Septicemia: See under specific organisms and diseases, as Typhoid; etc.
Septum, Nasal: See under Nose
Serositis, tuberculous polyserositis, 1110
Serum: See also Blood
Convalescent: See under Communicable Diseases; Measles; Meningitis; Scarlet Fever; Whooping Cough; etc.
Hypersensitiveness: See Anaphylaxis and Allergy
Immune: See also under Diphtheria; Meningitis; etc.
immune; production of passive immunity against influenza virus by introducing immune serums into respiratory tract, 653
Sickness: See Anaphylaxis and Allergy
Sex, factors influencing development of sexual attitudes and sexual awareness in children, *738
Hormones: See under Hormones; and under Ovary; Placenta; etc.
Intergrades: See under Hermaphroditism
Precocious Development: See under Adolescence
ratios in families with hemophilia, *1215
Shapiro, L. M.: Anemia of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
Shaw, D. R.: Production of passive immunity against influenza virus by introducing immune serum into respiratory tract, 653
Shaw, E. B.: Importance of sensitization mechanism in clinical phenomenon of allergy; possible cause and prevention, *581
Shepherd, M. L.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Shibley, G. S.: Active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
Shigella: See under Dysentery
Shock, Anaphylactic: See Anaphylaxis and Allergy
Shore, J.: Basal metabolism of tuberculous children; afebrile primary pulmonary tuberculosis, *119
Shuman, H. H.: Varicella in newborn, *564
Sigmoid: See also Colon; Intestines
rectosigmoidoscopic examinations in children's clinics, 628
Silverthorne, N.: Meningococcus and meningococcic infection, 1147
Simmonds' Disease: See under Pituitary Body
Simpson, R.: Hypoglycemia in 2 year old child with subtotal removal of pancreas, 1367
Sinus, Coronary: See under Heart
Sinuses, Nasal: See also Antrum; Frontal Sinus
acute sinusitis and acute rhinitis, 202
ephedrine in physiologic vehicle and lateral head low posture in treatment of nose and sinuses, 1328
sinusitis in children, 1329
Situs Inversus: See Viscera, transposition
Skeleton: See Bones
Skin, Abnormalities: See Ectodermal Defect
ascorbic acid content of diseased skin, 898
calcified and mummified epitheliomas of skin in children, 897
cutaneous manifestations of acute exanthematous diseases, 188
cutaneous manifestations of tularemia, 864
Diseases: See also Dermatitis; and under names of diseases, as Eczema; Herpes; etc.
diseases; anasarca due to nephrosis and intercurrent erysipeloid cutaneous lesions, 640
diseases; cutaneous lesions due to vitamin A deficiency in children, 602
diseases in children, 220
effects of high humidity on skin temperature at cool and warm conditions, 1348
multiple cutaneous abscess with recurrence in nursling, 1133
Pigmentation: See Pigmentation
Reactions: See Anaphylaxis and Allergy; etc.
studies in immunity; spontaneous changes in Dick and Schick reactions of children, *734
Skull: See Cranium
Sleep, effect on insensible perspiration in infants and children, *82
Smallpox, children vaccinated with antivariolar vaccine cultured in vitro, 613
combination of vaccinia with antivaccinial serum, 612
cultivation of vaccinia virus with reticuloendothelial cells, 610
experimental transformation of variola to vaccinia, 405
problems relating to vaccination of newborn, 860
varicella in newborn, *564
Smell, persistent anosmia following zinc sulfate nasal spraying, 201
Smith, C. A.: Atmosphere of reduced nitrogen with respiratory obstruction, 1351
Smith, C. E.: Acute coccidioidomycosis with erythema nodosum in children, 902

Smith, C. H.: Treatment of acute stomatitis, 907
Smyth, F. S.: Comparative studies on use of standardized protamine zinc and crystalline insulin, 919
Snell, D.: Rate of appearance of ossification centers from birth to age of 5 years, *949
Snelling, C. E.: Vitamin C requirements of infants, 1140
Social Security Act, maternal and child health services under Social Security Act, 1100
Societies, American Board of Pediatrics, examinations, 595, 923
American Board of Pediatrics, physicians certified by, 394
American Medical Association, meeting of Section on Pediatrics, 1371
American Orthopsychiatric Association, meeting, 1371
foreign, directory of, 230, 450, 684, 924, 1150, 1374
International Congress of Pediatrics, postponement of meeting, 1374
international, directory of, 230, 450, 684, 924, 1150, 1374
local, directory of, 233, 453, 687, 927, 1153, 1377
National Association for Nursery Education, biennial conference, 595
national, directory of, 231, 451, 685, 925, 1151, 1375
pediatric, directory of, 230, 450, 684, 924, 1150, 1374
proceedings of German Pediatric Society, 900
sectional, directory of, 231, 451, 685, 925, 1151, 1375
state, directory of, 232, 452, 686, 926, 1152, 1376
Society Transactions:
American Pediatric Society, 653, 902
Canadian Society for Study of Diseases of Children, 1137
Chicago Pediatric Society, 444
University of Michigan Pediatric and Infectious Disease Society, 1350
Sodium Citrate: See Angioma
Mandelate: See Urinary Tract, infections
Solis-Cohen, L.: Sporadic congenital goiter causing death of infant, *1067
Soluseptasine: See Meningitis; Sulfanilamide and Sulfanilamide Derivatives
Sontag, L. W.: Rate of appearance of ossification centers from birth to age of 5 years, *949
Souders, H. J.: Effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
Influence of fluid and of evaporated milk on mineral and nitrogen metabolism of growing children, *529
Sounds, effect of supersonic waves on virus of poliomyelitis, 409
Soybean, roasted, in infant feeding, 851
Spalding, J. E.: Diagnosis of trichinosis, *129
Spalding Sign: See Fetus, death of
Spasm: See also Chorea; Convulsions; Epilepsy; etc.
flying fetus, 857
Spasmophilia: See also Tetany
treatment with dihydrotachysterol, 602
Specialists, Medical, Directory of, 1090
Speech: See Language
Spina Bifida, congenital valves of posterior urethra, 428
roentgenologic aspects of spina bifida occulta, 1128
treatment of paradoxical incontinence of urine associated with spina bifida, 211
Spinal Cord: See also Meninges; Nervous System; etc.
Spinal Cord—Continued
congenital absence of abdominal musculature; associated changes in genitourinary tract and in spinal cord, *339
study and treatment of heredofamilial diseases with predominant lesions in, 632
Spinal Fluid: See Cerebrospinal Fluid
Spinal Puncture: See also Cerebrospinal Fluid
two hundred lumbar punctures of single patient, 1335
Spine, abnormalities; brevicollis, 184
abnormalities; Klippel-Feil syndrome, 218, 645
abnormalities; Klippel-Feil syndrome with cardiac hypertrophy in newborn infant, 186
abnormalities; typical case of malformation of fifth lumbar vertebra with roentgenogram and photograph, 892
changes in bodies of spinal vertebrae due to tetanus, 1320
curvature; management of threatened thoracogenic spinal curvature, 224
curvature; physiological scoliosis, 643
hemangioma of vertebral column, 436
hourglass tumors in children; review of literature and report of case, *131
hydatid cyst of spinal canal successfully treated by operation, 425
roentgenographic appearance of notches in child's vertebral bodies, 1344
uselessness of osteosynthesis in most cases of Pott's disease in children, 1109
Splenomegaly: See also Anemia, splenic
diagnosis of azar, 412
experimental studies on treatment of malarial splenomegalies by method of Ascoli, 1108
Sprue, Nontropical: See Celiac Disease
Sputum, contacts with sputum positive tuberculous cases, 1322
isolation of Bacillus tuberculosis from sputum, 868
new method of obtaining sputum from infants and children thought to have pneumococci in nasopharynx, 1139
Squint: See Strabismus
Stains and Staining, cytologic studies on rheumatic fever, 191
moth cerase and tubercle bacilli, 619
Staphylococci, bronchopulmonary staphylococcic infection of newborn; serotherapy, then vaccine therapy; cure, 608
diphtheritic or staphylococcic croup and simultaneous pneumococcic pneumonia, *817
empyema due to Staphylococcus aureus in infant 2 months old; treatment with staphylococcus anatoxin; cure, 626
infections at Singapore, 1318
lung diseases; pulmonary tuberculosis, mild bronchiectasis, staphylococcal empyema and diaphragmatic hernia into thorax, 420
primary bronchogenic staphylococcic pneumonia, 205
relapses in osteomyelitis of staphylococcic origin, 891
source of staphylococcal infection in acute osteomyelitis, 433
stability and antigenicity of staphylococcic toxoid, 597
staphylococcic anatoxin and therapy of staphylococcic infections in general, 616
staphylococcus septicemia, 411
transfusions in pediatric orthopedics, 1134
Starch, digestibility of raw potato starch in man, 849
Starvation: See Hunger
Status Asthmaticus: See Asthma
Dysmyelinatus: See under Nervous System
Steatorrhea: See Celiac Disease
Stein, J. L.: Meningitis caused by pneumococcus type III; observations on sulfanilamide therapy, *274
Steinbach, M.: Sporadic congenital goiter causing death of infant, *1067

Steiner, M. M.: Meningitis caused by pneumococcus type III; observations on sulfanilamide therapy, *274
Stepan, C. E.: Hyperpyrexia following operation for cleft lip or palate, *320
Sterilization, air contamination and air sterilization, *1023
Stern, M. E.: Diphtheritic or staphylococcic croup and simultaneous pneumococcic pneumonia, *817
Sternum, Puncture: See Bones, marrow
Stewart, A.: New creatinine standard for basal metabolism and its clinical application, *506
Stewart, C. A.: Tuberculosis in children less than 6 years of age, *92
Still's Disease: See Arthritis deformans
Stillbirths: See Infant Mortality
Stokes, J., Jr.: Production of passive immunity against influenza virus by introducing immune serums into respiratory tract, 653
Stokes-Adams' Disease: See Heart, block
Stomach: See also Gastrointestinal Tract; Pylorus; etc.
congenital pyloric stenosis with cardiostenosis in child aged 4 days, 1117
culture of tubercle bacilli in stomach washings of children, 620
gastroscopic observation of effect of aspirin and certain other substances on stomach, 209
rupture in newborn, 185
Ulcer: See Peptic Ulcer
Stomatitis, acute, treatment of, 907
esophagitis in infancy, 209
gangrenous; noma with recovery following administration of quinine, 423
herpetic; enanthem, 907
impetiginous, 422
Vincent's: See Angina, Vincent's
Stools: See Feces
Stovarsol: See Acetarsone
Strabismus, orthoptic treatment of concomitant convergent strabismus, 1131
some aspects of, 436
surgical treatment, 220
Straining and coughing, effect on blood pressure, *115
Streptococci: See also Erysipelas; Meningitis; Scarlet Fever; etc.
bactericidal effect of sulfanilamide on beta hemolytic streptococci in vitro, 177
diseases, 616
hemolytic, in etiology of rheumatic fever and rheumatoid arthritis, 192
otitic meningitis due to Streptococcus hemolyticus; operation and recovery, 630
para-aminobenzenesulfonamide and its derivatives in treatment of beta hemolytic streptococcus infections of middle ear and mastoid; report of 6 cases in children, 202
precipitin test applied to Melbourne hemolytic streptococci, 848
purulent meningitis cured by sulfanilamide; discussion on nature and possible relapse of streptococcic meningitis, 627
streptococcal peritonitis with septicemia, 880
studies on hemolytic streptococci from human sources; cultural characteristics of potentially virulent strains, 847
suppurative streptococcic lymphadenitis following diphtheria in brothers, 864
treatment of purulent streptococcic meningitis by sulfanilamide, 631
Streukens, T.: Tuberculosis in children less than 6 years of age, *92
Strümpell's Disease: See Paralysis, spastic
Struthers, R. R.: In vitro experiments on effect of added blood serum and blood plasma on sedimentation rate, 1137
Strychnine poisoning in 16 month old infant, 1368
Stuart, H. C.: Roentgen studies of patterns of growth of muscle and other tissues in young children, 680
Subdural Space: See Meninges
Sucrose, comparative values of various carbohydrates used in infant feeding, 178
Therapy: See Asthma
Sugar cane, new carbohydrate for prevention of nutritional anemia in infants; preliminary report, *45
Sugars: See Dextrose; etc.
in Blood: See Blood, sugar
in Urine: See Diabetes Mellitus
Sulfanilamide and Sulfanilamide Derivatives, 226, 1348
bactericidal effect of sulfanilamide on beta hemolytic streptococci in vitro, 177
chemotherapy of bacterial infections, 848
comparison of different methods in use for determination of sulfanilamide in tissues and fluids of body, 1364
delayed toxic reactions to sulfanilamide, 1347
eruptive fever (dengue, measles, German measles?) in reality complex toxic drug dermatitis (pentavalent arsenic and sulfanilamide derivative), 650
granulocytopenia associated with sulfanilamide therapy, 224
mechanism of action of sulfanilamide, 650
mode of action, clinical use and toxic manifestations of sulfanilamide, 1135
neutropenia following sulfanilamide; report of case, 224
paraaminobenzenesulfonamide [sulfanilamide], 900
physiological effects of sulfanilamide; methemoglobin formation and its control, 1348
placental transfer of sulfanilamide, 184
severe hemolytic anemia due to sulfanilamide, 1111
study of action of sulfanilamide, 442
sulfanilamides in tertian malaria, 192
Therapy: See Chorea; Endocarditis; Erysipelas; Gonorrhea; Malaria; Mastoiditis; Meningococci; Meningitis; Rheumatic Fever; Scarlet Fever; Urinary Tract; Whooping Cough; etc.
use of sulfanilamide in some clinical conditions, 1349
Sulfapyridine, Therapy: See Whooping Cough
Sulfur: See also Cystine and Cysteine
and protein metabolism of naturally and artificially fed infants, 1305
metabolism of early infancy, *37
Sunlight: See Ultraviolet Rays
Sunspots: See Meteorology
Supersonic Waves: See Sounds
Suprarenal Preparations: See Adrenal Preparations
Suprarenals: See Adrenals
Surgery, pediatrist and surgeon, 225
plastic, in children; medical and psychologic aspects of deformity, 441
Swanson, W. W.: Sulfur metabolism of early infancy, *37
Syncope, 1359
Synostosis, congenital bilateral radio-ulnar synostosis, *1259
Syphilis: See also under names of organs and regions
brittleness of bones in syphilitic nursling, 414
children of syphilitic mothers and adoption, 198
colloidal carbon reaction of spinal fluid, 1110
congenital; involuntary defecation in 9 year old girl with congenital syphilis, 870
congenital, mapharsen in treatment, 618
congenital; ocular and dental malformations and disturbances of personality in 2 brothers with hereditary syphilis, 869

Syphilis—Continued
congenital, ocular manifestations of, 219
congenital, when to treat, 869
cytologic studies on rheumatic fever, 191
fecal incontinence in children; 20 cases, 422
generalized osseous dystrophy, 644
Hereditary: See Syphilis, congenital
microtest of, 620
pentavalent arsenic in treatment, 869
pseudoleukemic splenic anemia in syphilitic infant, 869
serologic reactions in syphilis and malaria in infancy, 617
serous apoplexy in children in course of treatment with arsenobenzenes, 631
spinal fluid of syphilitic children, 414
Syringomyelia, pathogenesis; case illustrating process of cavity formation from embryonic cell rests, 1118

Tabes Dorsalis in children, 1324
Tachycardia: See under Heart
Talbot, N. B.: New creatinine standard for basal metabolism and its clinical application, *506
Tanning: See Pigmentation, cutaneous
Tarsus, cranio-carpo-tarsal dystrophy of ? undescribed type; 2 cases, 217
Tay-Sachs' Disease: See Idiocy, amaurotic
Teeth, defect of parallelism between osseous growth and dental development considered as factor in etiology of dentofacial malformations, 223
diet and its relation to dental disease, 1134
experimental scurvy of guinea pig, 603
factors in control of dental caries of children, 920
familial cleidocranial dysostosis (cleidal dysostosis); preosseous and dentinal dystrophy, *786
heredity in relation to size, 222
hydroa aestivale in congenital porphyria, 1132
metabolism in dental decay and prophylaxis against caries, 1135
nutritional factors in dental development, 222
ocular and dental malformations and disturbances of personality in 2 brothers with hereditary syphilis, 869
prevention of dental caries, 1135
preventive dentistry in preschool period and during fetal life, 1135
relation of diet to oral health, 222
Telangiectasia, histopathology of Osler's disease (hereditary hemorrhagic telangiectasia), 440
Temperature: See also Fever; Heat; etc.
effects of high humidity on skin temperature at cool and warm conditions, 1348
noninfectious elevation among adolescents, 1349
Temporal Bone, changes in rickets and in "renal rickets," 1098
petrosquamous osteomyelitis in infants, 1329
problems of suppurative petrositis and its surgical treatment together with outline of prophylaxis and treatment of otitic meningitis, 203
progressive facial palsy produced by intratemporal epidermoids, 900
Teratoma of neck of fetus with acute hydramnios, 1308
Testicles, Internal Secretion of: See Androgens; Gonads; Hormones; etc.
undescended, diagnosis of, 215
undescended; hormone treatment of imperfect descent of testis; further observations, 429
Testosterone Propionate: See Androgens
Tetanus antitoxin, 615
changes in bodies of spinal vertebrae due to, 1320
comparative studies on purification of tetanus and diphtheria toxins, 847
in children, 617
Tetanus—Continued
neuritis caused by prophylactic vaccination against tetanus, 194
reports of few cases of tetanus treated with antitetanic serum and magnesium sulphate, 866
therapy of, 1322
two cases, 617
Tetany: See also Spasmophilia
actions of crystalline vitamin D_3 (calciferol) in chronic parathyroid tetany, 600
chronic idiopathic parathyroid tetany, 430
Tetralogy of Fallot: See Heart, abnormalities
Thalhimer, W.: Immunologic properties of scarlatina convalescent serum, *1039; correction, 1372
Thaysen-Gee Disease: See Celiac Disease
Thelander, H. E.: Importance of sensitization mechanism in clinical phenomenon of allergy; possible cause and prevention, *581
Perforation of gastrointestinal tract of newborn infant, *371
Thermotherapy: See under names of diseases
Thiamin: See Vitamins, B
Thinness, boy aged 4 years weighing 10 pounds, 850
loss of weight caused by protozoa, 1319
Thirst, effect of thirst and salt deficiency on water depots of organism, 1304
Thomsen's Disease: See Myotonia congenita
Thorax: See also Heart; Lungs; Mediastinum; Pleura; etc.
funnel chest, 1130
lung diseases; pulmonary tuberculosis, mild bronchiectasis, staphylococcal empyema and diaphragmatic hernia into thorax, 420
management of threatened thoracogenic spinal curvature, 224
neuroblastoma of left adrenal with intrathoracic metastases, 432
thoracic deformity, 890
unusual fatal perforating wound of chest, *1278
Throat: See also Larynx; Tonsils; etc.
demonstration of pneumococci in throats of normal infants and of infants with pneumonia or infection of upper respiratory tract, 1330
Thrombocytes: See Blood, platelets
Thrombopenia: See under Diathesis, hemorrhagic; Purpura haemorrhagica
Thrombosis: See also Embolism
of splenic vein (so-called splenic venous stenosis), 417
Thrush: See Stomatitis
Thumb Sucking: See Finger Sucking
Thymus, 430
and thymicolymphatic constitution; clinical aspects as seen by obstetrician, 1358
and thymicolymphatic constitution; clinical aspects as seen by otolaryngologist, 1359
and thymicolymphatic constitution; introductory remarks, 1357
and thymicolymphatic constitution; roentgen findings, 1360
certain iodine-reducing substances of thymus extract, 889
effect of antuitrin S on young albino rat, 641
effect of castration on weight of thymus in adult albino rat, 1343
effect of injections of extract of thymus (Hanson) on growth of albino rat, 1342
effect of prepuberal castration of female albino rat on thymus and lymph glands, 1342
effect of thyroidectomy and of adrenalectomy on thymus of castrated white rat, 1342
effects of thymectomy on growth of albino rats, 1342
end-results of irradiation of thymus gland in 24 normal infants and children, 888
hematoneurologic syndrome with disturbance of dextrose metabolism from thymicopancreatic dysfunction, 642

Thymus—Continued
hyperplasia, radiology's responsibility in diagnosis and treatment, 431
hypertrophy in course of acute leukemias of childhood, 1326
hypertrophy, pneumographic examination of anterior mediastinum in diagnosis of atypical forms of, 891
Klippel-Feil syndrome with cardiac hypertrophy in newborn infant, 186
studies of some changes in gonads and pituitary following its destruction by roentgen irradiation, 890
thymic asthma, 1128
tumors and their association with myasthenia, 642
Thyroid: See also Goiter; Parathyroid
acute suppurative thyroiditis during childhood, *837
effect of thyroidectomy and of adrenalectomy on thymus of castrated white rat, 1342
effect of vitamin B_1 anid vitamin B_2 complex on loss of weight produced in rats by experimental hyperthyroidism, 1306
effect on growth rate of thyroparathyroidectomy in newborn rats and of subsequent administration of thyroid, parathyroid and anterior hypophysis, 640
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on calcium retention, *1186
factors affecting retention of nitrogen and calcium in period of growth; effect of thyroid on nitrogen retention, *965
function of thyroid and pituitary in mongolism with report on blood groups of American mongoloid defectives and comments on determination of cholesterol, *1240
Hypothyroidism: See also Cretinism; Myxedema
hypothyroidism in children, 1353
oral administration of hormonal proteins, 888
renal lesion in hyperparathyroidism, 1127
role of iodine (potassium iodide) in experimental hyperthyreoidism, 642
studies in mongolism, 1338
thyrotoxicosis in child aged 6 years; thyroidectomy, 1128
vitamin A deficiency in disease of thyroid gland; its detection by dark adaptation, 1127
Thyroidectomy: See Thyroid
Thyrotoxicosis: See Thyroid
Thyroxine, calorigenic potency of free thyroxine by mouth, 1125
Tibia, diagnosis of kala-azar by search for parasite in bone marrow (puncture of metaphysial region of tibia), 411
Tic in childhood, 424
Tick Fevers: See Rocky Mountain Spotted Fever; Tularemia
Tisdall, F. F.: Air contamination and air sterilization, *1023
Tissue, Adipose: See Fat; Obesity
comparison of different methods in use for determination of sulfanilamide in tissues and fluids of body, 1364
roentgen studies of patterns of growth of muscle and other tissues in young children, 680
Toes: See Fingers and Toes
Tongue, congenital hemihypertrophy of, 403
Tonsillectomy, acute anterior poliomyelitis following tonsillectomy and adenoidectomy with reference to bulbar form, 1105
and nasal diphtheria, 864
hemorrhage following, 1114
in presence of acute peritonsillar abscess, 874
incidence in school children, 203
local anesthesia in children, 1134
occurrence of blood in air passages after tonsillectomy, 875
Tonsillitis: See under Tonsils
Tonsils, Abscess: See under Abscess
biologic significance of tonsils and adenoids and other external lymphoid masses, 1113
incidence of tuberculous infection in surgically removed tonsils, 196
roentgen treatment of, 626
tuberculosis of, 200
Toomey, J. A.: Second attacks of experimental poliomyelitis in Macacus rhesus monkeys; immunity or lack of immunity to Philadelphia 1932 strain of virus, *41
Topper, A.: Basal metabolism of tuberculous children; afebrile primary pulmonary tuberculosis, *119
Basal metabolism of tuberculous children; children with pneumothorax, *957
Basal metabolism of tuberculous children; tuberculosis of bone, *778
Insensible perspiration in children; influence of alterations in vegetative nervous system induced by atropine, pilocarpine and epinephrine, *71
Tortone, J.: Tuberculosis in children less than 6 years of age, *92
Torula, meningoencephalitis due to Cryptococcus meningitidis (Torula histolytica), 1119
Tower Head: See Acrocephaly
Towsley, H. A.: Cord blood serum for prophylaxis and modification of measles, 1357
Toxemia, toxic syndrome in children, 652
Toxin and Antitoxin: See under Diphtheria; Staphylococci; Tetanus; etc.
Toxoid: See under Diphtheria; Scarlet Fever; Tetanus; etc.
Toxoplasmosis, granulomatous encephalomyelitis due to protozoan (Toxoplasma or Encephalitozoon); identification of case from literature, 1335
Trachea, compression of, 1359
Transfusion: See Blood, transfusion
Tremor: See also Paralysis, agitans
congenital heredofamilial disease, consisting of rhythmic tremor of head, eyes and upper extremities, 631
Trench Mouth: See Angina, Vincent's
Trichinosis, diagnosis of, *129
treatment with calcium, 1319
Trichocephaliasis, large doses of iron in treatment of trichocephalus infection, 617
Trichomonas, cultivation of Trichomonas foetus in chick embryo, 1302
Trichophytin: See Trichophytosis
Trichophytosis, trichophytin test; its value as diagnostic aid, 1346
Tubercle Bacilli: See also Sputum
and moth cerase, 619
culture in stomach washings of children, 620
phagocytosis in children (Otani's method), 620
studies on filtrable form, 620
Tuberculin: See also Tuberculosis
old tuberculin and purified protein derivative in tuberculin patch test; comparative study, *527
Tuberculoma, cerebral, 212
intracranial, 212
Tuberculosis: See also Tubercle Bacilli; and under names of organs, regions and diseases, as Abdomen; Bones; Endocarditis; Lymph Nodes; Meningitis; Muscles; Palate; Tonsils; etc.
allergy of infants vaccinated with B C G, 196
antituberculous vaccination with B C G, 1322
basal metabolism of tuberculous children; children with pneumothorax, *957
basal metabolism of tuberculous children; tuberculosis of bone, *778
care of tomorrow, 1111
contacts with sputum positive tuberculous cases, 1322
culture of bacillus tuberculosis from blood by Massalski method, 198

Tuberculosis—Continued
culture of tubercle bacilli in stomach washings of children, 620
death from hemoptysis occurring in infant of 9 months, 1322
end results in patients with severe tuberculosis of bone and with large tuberculous sequestrae, 1323
fate of patients suffering from phlyctenular disease with reference to tuberculosis, 437
human immunity against tuberculosis, 870
immunization against, 195
immunologic characteristics of different types of tuberculin reactors, 867
in American schools and colleges, 868
in children less than 6 years of age, *92
incidence of tuberculous infection in surgically removed tonsils, 196
infantile, contagiousness of, 1324
influence of pure vitamin preparations on course in guinea pigs, 870
intracranial tuberculoma, 212
intracutaneous tuberculin test of Mantoux; method employing single injection, 619
irritation and toxic susceptibility in course of rheumatic infection, 412
isolation of Bacillus tuberculosis from sputum, 868
juvenile, problems of prevention and treatment in, 1324
late results in 9 children vaccinated with B C G, 869
lung diseases; pulmonary tuberculosis, mild bronchiectasis, staphylococcal empyema and diaphragmatic hernia into thorax, 420
miliary, congenital origin of, 1109
miliary; pseudomiliary lesions in lungs, 627
multiple puncture method of B C G vaccination, 196
old tuberculin and purified protein derivative in tuberculin patch test; comparative study, *527
primary, pleurisy as allergic manifestation of, 414
problem in infants in relation to modern investigations of ultravirus, 1324
prognosis in infants and children, 414
pulmonary; artificial pneumothorax in children, 868
pulmonary; basal metabolism of tuberculous children; afebrile primary pulmonary tuberculosis, *119
pulmonary; cerebral paratuberculosis, 413
pulmonary, contralateral pneumothorax in treatment, 197
pulmonary; right diaphragmatic hernia, 422
pulmonary; syndrome of Löffler, 878
pulmonary, types of; creeping tuberculosis, 1322
reaction of children vaccinated with B C G to tuberculous infection, 413
revision of immunopathologic concept, 413
roentgenographic appearance of miliary distribution at onset of ambulatory pulmonary tuberculosis, 1109
sensitivity to tuberculin in rheumatic disorders, 412
sequence of roentgen evidence of tuberculosis and cutaneous sensitivity to tuberculin; study of infants, *799
tuberculin patch tests in children, 1109
tuberculous endocarditis of pulmonary valve, 871
tuberculous polyserositis, 1110
undesirable tuberculin reactions, 415
Yoshida's test in regard to active tuberculosis, 1110
Tularemia, cutaneous manifestations of, 864
Tumors: See also Angioma; Cancer; Glioma; Sarcoma; etc.; and under names of organs and regions, as Bones; Brain; Cerebellum; Kidneys; etc.
embryonal sympathetic tumor in child, 882
Tuna Liver Oil: See under Oil
Twins, anemia of newborn in 1 identical twin and universal edema of newborn in other with survival in both cases, *1050
disease in, 184
myatonia congenita in identical twins, 435
Typhoid at Volendam, Netherlands, 1321
carriers, 1321
in infant 5 months old, 412
local and peripheral leukocyte counts in certain infectious diseases, 1315
outbreak of milk-borne typhoid, 193
septicemia due to Diplococcus crassus, simulating typhoid, 412

Ulcers: See under names of organs and regions
Peptic: See Peptic Ulcer
Ulna, congenital bilateral radio-ulnar synostosis, *1259
subperiosteal resection of, 442
vitamin C deficiency and periostitis of both ulnae? scurvy, 1097
Ultraviolet Rays: See also under names of organs and diseases
air contamination and air sterilization, *1023
inhalation therapy and ultraviolet rays in preventive pediatrics, 1349
lead poisoning produced by irradiated lead, 443
of diffuse daylight and its therapeutic utilization, 397
some experiences with mercury vapor lamp, 1132
tanning from ultraviolet irradiation brought out by blood transfusion, *1047
Umbilical Cord, cord blood serum for prophylaxis and modification of measles, 1357
umbilical discharges in infant, 609
Umbilicus, navel colic, 629
Urea content of brain of newborn, 1311
Urease reaction of Brucella abortus, 597
Ureters: See also Urinary Tract
atresia of ureter and agenesis of kidney, 1124
coexistence of abnormality of vesical neck and bilateral megaureter in child 8 years old; multiple operations; functional cure, 639
duplication of ureter and paravaginal cyst, 1125
expectancy of life after ureteral transplantation, 428
Urethra, congenital valves of posterior urethra, 428
Urinary Tract: See also Genitals; Genitourinary Tract; Kidneys; Ureters; etc.
comparison of organic acids and sulfanilamide as urinary antiseptics, 429
Infections: See also Bladder, inflammation; Nephritis; etc.
infections; graver types of colon bacillus infection in infant's urinary tract, 429
infections in infancy and childhood; diagnosis and treatment, 428
infections; treatment of Bacillus coli infection with mandelic acid, 1341
infections with Bacillus dysenteriae, *830
urologic conditions in childhood, 215
Urination, excessive; pseudohermaphroditism, adiposity, polyuria and hyperglycemia; infundibulotuberian syndrome, 630
incontinence; enuresis and other factors in normal and in psychotic persons; comparative study of incidence and intercorrelations, 428
incontinence; treatment of paradoxical incontinence of urine associated with spina bifida, 211
Urine: See also Diuresis and Diuretics; Urination; under names of diseases; etc.
Albumin: See Albuminuria
creatine; significance of creatinuria in progressive muscular dystrophy, 892
excretion of vitamin C in certain diseases of children, 1306

Urine—Continued
Hormones in: See under Pregnancy, urine in
metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
passage of pigment of red beet into urine of child, 639
Ustilago, further report on corn rust poisoning in childhood, 1136
Uterus, inclusion blennorrhea, 1102
Uveoparotid Fever, "neuro-uveo-parotitis," 632

Vaccination: See Immunity; and under names of various diseases, as Smallpox, vaccination; Typhoid, immunity; etc.
Encephalitis Following: See under Encephalitis
Vaccine Therapy: See under Diphtheria; Respiratory Tract; Typhoid; Whooping Cough; etc.
Vaccinia: See Smallpox
Vagina, diagnosis of hematocolpos, 1341
duplication of ureter and paravaginal cyst, 1125
hematocolpos with congenital imperforate hymen, 1124
treatment of gonorrheal vaginitis with estrogen, 217
Vaginitis, Gonococcic: See Gonorrhea
Vagotonia: See Nerves, vagus
Varicella: See Chickenpox
Variola: See Smallpox
Vegetables, mode of action in feeding of infants, 395
nitrogen metabolism and utilization of foodstuffs in children receiving diets containing vegetable protein, 851
studies on local products of Hunan; vitamin C content of "vegetable water," 397
vitamin C content of canned vegetables, 1307
Veins: See also Cardiovascular System; Embolism; Thrombosis; etc.
pulmonary, anomalous drainage into coronary sinus, *354
splenic, stenosis in childhood, 622
thrombosis of splenic vein (so-called splenic venous stenosis), 417
Venereal Diseases: See Gonorrhea; Syphilis
Venesection, capillary bronchitis; its treatment by venoclysis and blood transfusion, 878
Venoclysis: See Venesection
Ventriculography: See under Brain, roentgenography
Verruca, treatment of, 898
Vertebrae: See Spine
Vestibular Apparatus: See Ear
Vincent's Angina: See Angina, Vincent's
Viosterol, histologic study of hypervitaminosis D; relative toxicity of vitamin D of irradiated ergosterol and tuna liver oil, 1095
Viruses: See also Herpes; Rabies; Tubercle Bacilli; etc.
German measles as virus disease, 409
problem of tuberculosis in infants in relation to modern investigations of ultravirus, 1324
studies on inclusion blennorrhea, 1102
Viscera, transposition, genetics of, 606
Vision: See Blindness; Eyes; etc.
Vital Capacity, basal metabolism of tuberculous children; children with pneumothorax, *957
Vital Statistics, death rate in children 1 to 4 years old in Netherlands, 605
Vitamins: See also Cod Liver Oil; Deficiency Diseases; Diet and Dietetics; Food; Milk; Rickets; etc.
A: See also Carotene; Cod Liver Oil; Xerophthalmia
A deficiency, cutaneous lesions due to, 602
A deficiency in disease of thyroid gland; its detection by dark adaptation, 1127
A deficiency in man; resolution of cutaneous lesions following parenteral administration of carotene, 183
A; diet and rate of depletion of hepatic vitamin A, 1096

Vitamins—Continued
A; efficacy of parenteral vitamin A administration in vitamin A deficiency; clinical study of 9 cases, 183
A; further studies on Werlhof's morbus maculosus; hitherto unknown fat-soluble T factor which causes thrombocytosis, 873
A; hypervitaminosis A, 853
A research and its clinical applications in pediatrics, 181
A, studies of, 665
avitaminotic edema of infancy, 440
B: See also Beriberi; Pellagra
B; clinical significance and estimation of blood vitamin B_1, 852
B; distribution of vitamin B_1 (thiamin) in meat and meat products, 1096
B; effect of vitamin B_1 and vitamin B_2 complex on loss of weight produced in rats by experimental hyperthyroidism, 1306
B; effect of yeast and of thiamin on production of low iodine goiter, 641
B; experimental postdiphtheritic paralysis and vitamin B_1, 1107
B fractions and insulin tolerance in albino rat, 1305
B; hepatic injury on nutritional basis in rats, 920
B; new knowledge on relation of liver function to vitamin B_1, 855
B; note on vitamin B_1—sparing action of fat, 1096
B; prevention of dental caries, 1135
B; vitamin B_1 and glycemia, 1307
C: See also Scurvy
C, administration in form of ascorbic acid and of fruit juice, 856
C and acute infections of upper respiratory tract, 1329
C and rheumatic infection, 615
C and scurvy, 853
C; ascorbic acid and complement function, 601
C; ascorbic acid content of diseased skin, 898
C; ascorbic acid metabolism of very small children and those of school age, 602
C; ascorbic acid requirements in early infancy, 182
C; clinical test for determining vitamin C deficiency, 181
C; complementing activity and ascorbic acid content of guinea pig serums following ether anesthesia, 601
C content of canned vegetables, 1307
C content of spinal fluid, 1336
C deficiency and periostitis of both ulnae? scurvy, 1097
C deficiency, determination of, 182
C; determination of ascorbic acid, 667
C; effect of ascorbic acid on constitution of complement, 601
C, effect on Schick reaction, 603
C in spinal fluid during hunger, 603
C, investigations of vitamin C standard in healthy children and in children suffering from gingivitis, 398
C; lack of correlation of capillary fragility with vitamin C content of blood, spinal fluid and urine, 198
C nutrition and susceptibility to diphtheria as possible factors in epidemiology of poliomyelitis, 1314
C nutrition in artificial fever, 1305
C, rectal absorption of, 397
C; relation of leukocytosis to ascorbic acid requirements, 852
C, requirements in man, 601
C requirements of infants, 1140
C; studies on Hunan local food products; chemical composition and vitamin C content of Hunan citrus fruits, 604

Vitamins—Continued
C, studies on local products of Hunan; vitamin C content of "vegetable water," 397
C; study of breed and seasonal variations in ascorbic acid content of certified milk from Guernseys and Holsteins, 1096
C therapy for hemorrhagic diatheses, 1327
C; treatment of nutritional disorders in infants with ascorbic acid, 854
C, urinary excretion in certain diseases of children, 1306
C; verification of tests of hypovitaminosis C in children of school age, 1306
D: See also Cod Liver Oil; Rickets; Viosterol
D; actions of crystalline vitamin D_2 (calciferol) in chronic parathyroid tetany, 600
D; comparison of effects of A. T. 10 (dihydrotachysterol) and vitamin D on calcium and phosphorus metabolism in hypoparathyroidism, 600
D; effect of irradiated milks on storage of nitrogen and acid-base minerals in children, *753
D, effect on calcium and phosphorus metabolism; studies on 4 patients, 600
D; histologic study of hypervitaminosis D; relative toxicity of vitamin D of irradiated ergosterol and tuna liver oil, 1095
D intoxication in infancy, 666, 1142
D; reappraisal of vitamin D milks, 178
D; use of vitamin D preparations in prevention and treatment of disease, 181
Deficiency: See also Beriberi; Deficiency Diseases; Rickets; Scurvy; etc.; and under specific vitamins
deficiencies, uptake of tissues in, 1095
E; experiments with wheat germ oil (vitamin E) in nutrition of premature infants, 1102
E; its influence on lactation of mothers and growth of their children, 855
experimental investigation on relationship of vitamins to infection, 602
influence of pure vitamin preparations on course of tuberculosis in guinea pigs, 870
influence of vitamin B_1 and vitamin C on anaphylactic shock, 855
influence of vitamins C, B_1 and B_2 on cholesterol content of blood in children, 397
K, inactivation by light, 1306
K; prothrombin in chickens, 870
newer aspects of nervous disorders in avitaminosis, 852
newer developments in child nutrition, 850
recent conceptions of vitamin activity, 855
review of differential diagnosis of osteopathies due to hormonal causes, avitaminosis and kidney disease, 1345
vitamin therapy, 853
Vitreous Humor, hitherto unknown hereditary ocular disease (degeneratio hyaloidearetinalis hereditaria) observed in canton of Zurich; report of cases, 1131
Voice, dysphonia aspects of mongolian imbecility, 1338
Vollmer, H.: Old tuberculin and purified protein derivative in tuberculin patch test; comparative study, *527
Volpitto, P. P.: Accuracy of clinical determinations of blood pressure in children with values under normal and abnormal conditions, *102
Vomiting in children, 1332
Vulvovaginitis, Gonococcic: See Gonorrhea

Walifield, M. J.: Pleural manifestations of perforation of esophagus, *1261
Walthall, D.: Postlactation feeding, 1350
Wang, C. C.: Metabolic study of 5 children with nephrotic syndrome; fat and protein metabolism and creatinine and creatine output, *29
Ward, F. S.: Air contamination and air sterilization, *1023
Warts: See Verruca
Wassermann Reaction: See under Syphilis
Water, effect of thirst and salt deficiency on water depots of organism, 1304
Metabolism: See also Dehydration; Diuresis and Diuretics; Edema
metabolism; mineral and water metabolism in early infancy and its relation to problem of artificial feeding, 1095
phenol-contaminated waters and their physiological action, 177
studies in physiology of artificial fever; changes in blood volume and water balance, 1348
Watson, E. H.: Pituitary therapy and carbohydrate tolerance, 1369
Weaning: See Infant Feeding
Wechsler, I. S.: Erythroblastic (Cooley's) anemia and neurologic complications (status dysmyelinatus), *362
Weech, A. A.: Reaction of edematous patient to transfusion with serum or with acacia, 915
Weight: See Body, weight
Werdnig-Hoffmann Paralysis: See Atrophy, muscular
Werlhof's Disease: See Purpura haemorrhagica
Wheat Germ Oil: See Vitamins, E
Whooping Cough, active immunization against pertussis; final report on Cleveland immunizations of 1934-1935, *691
acute encephalitis of, 1106
attempt to increase resistance to pertussis in newborn infants by immunizing their mothers during pregnancy, 1104
Bacillus para-pertussis; species resembling both Bacillus pertussis and Bacillus bronchisepticus but identical with neither, 610
effect of hyperimmune human serum (lyophile) and of sulfapyridine on experimental murine pertussis, *1228
effect of hyperimmune human serum (lyophile) on humoral antibody titer in pertussis, *1234
experimental infection of mice with Haemophilus pertussis, 609
new phases of work on immunization and prophylaxis, 405
prophylactic use of pertussis vaccine (Sauer), 406
reaction following injection of pertussis vaccine—Sauer, 187
vaccine, 614
Wilbar, C. L., Jr.: New carbohydrate for prevention of nutritional anemia in infants; preliminary report, *45
Williams, W. E.: Vitamin D intoxication in infancy, 666, 1142
Wilms Tumor: See Kidneys, tumors
Wilson's Disease: See under Lenticular Nucleus
Wolarsky, W.: Infection of urinary tract with Bacillus dysenteriae, *830
Wold, M.: Effect of hyperimmune human serum (lyophile) and of sulfapyridine on experimental murine pertussis, *1128
Wolman, I. J.: Clinical and experimental studies of lipoid pneumonia, 654
Diagnosis of congenital cardiac defects in infancy; study of 32 cases with necropsies, *8
Woodbury, R. A.: Accuracy of clinical determinations of blood pressure in children with values under normal and abnormal conditions, *102
Worcester, J.: New creatinine standard for basal metabolism and its clinical application, *506
Worms: See Intestines, parasites; etc.
Wright, F. H.: Circulatory function in anemias of children; roentgenographic measurement of cardiac size, *250

Wrist, cranio-carpo-tarsal dystrophy of ? undescribed type; 2 cases, 217
deformities; Madelung's disease, 219
effect of dietary supplement on ossification of bones of wrist in institutional children; effect of cod liver oil supplement, *295

Xanthelasmatosis: See Xanthoma
Xanthochromia, biochemical studies of blood and spinal fluid in newborn, 1103
Xanthoma molluscifórme et generalisatum in infancy, 1347
tuberosum, congenital, 437
xanthelasmatosis in childhood, 1327
Xanthomatosis: See Anemia, splenic; Schüller-Christian Syndrome; Xanthoma
Xerophthalmia, anatomopathologic study in infants, 646
efficacy of parenteral vitamin A administration in vitamin A deficiency; clinical study of 9 cases, 183

Yannet, H.: Experimental study of cerebral changes in prolonged hypoglycemia, 918
Yeast, effect of yeast and of thiamin on production of low iodine goiter, 641
Yoshida's Test: See Tuberculosis

Ziegler, M. R.: Experimental studies concerning pathology of insulin convulsions, 918
Zinc, quantitative study, by means of spectrographic analysis, of zinc in nutrition, 1093
Sulfate: See under Poliomyelitis

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CONTENTS

PAGE

THE DISABLING DISEASES OF CHILDHOOD: THEIR CHARACTERISTICS AND MEDICAL CARE AS OBSERVED IN 500,000 CHILDREN IN EIGHTY-THREE CITIES CANVASSED IN THE NATIONAL HEALTH SURVEY OF 1935-1936. DOROTHY F. HOLLAND, PH.D., WASHINGTON, D. C. 1157

FACTORS AFFECTING RETENTION OF NITROGEN AND CALCIUM IN PERIOD OF GROWTH: II. EFFECT OF THYROID ON CALCIUM RETENTION. J. A. JOHNSTON, M.D., AND JOHN W. MAHONEY, M.D., DETROIT 1186

THE PLAY-INTERVIEW: A METHOD OF STUDYING CHILDREN'S ATTITUDES. JACOB H. CONN, M.D., BALTIMORE 1199

SEX RATIOS IN FAMILIES WITH HEMOPHILIA. MADGE THURLOW MACKLIN, M.D., LL.D., LONDON, ONTARIO, CANADA 1215

EFFECT OF HYPERIMMUNE HUMAN SERUM (LYOPHILE) AND OF SULFAPYRIDINE ON EXPERIMENTAL MURINE PERTUSSIS. W. L. BRADFORD, M.D., AND MARY WOLD, B.A., ROCHESTER, N. Y. 1228

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FUNCTION OF THE THYROID AND THE PITUITARY IN MONGOLISM, WITH A REPORT ON THE BLOOD GROUPS OF AMERICAN MONGOLOID DEFECTIVES AND COMMENTS ON THE DETERMINATION OF CHOLESTEROL. CLEMENS E. BENDA, M.D., AND EMILY MAY BIXBY, PH.D., WRENTHAM, MASS. .. 1240

CASE REPORTS:

SEVERE HEMORRHAGE SECONDARY TO RETROPHARYNGEAL ABSCESS. FRED Z. HAVENS, M.D., ROCHESTER, MINN. 1256

CONGENITAL BILATERAL RADIO-ULNAR SYNOSTOSIS. ALMON R. CROSS, M.D., DURHAM, N. C. 1259

PLEURAL MANIFESTATIONS OF PERFORATION OF THE ESOPHAGUS. MARK J. WALLFIELD, M.D., BROOKLYN 1261

PNEUMONIA COMPLICATED BY AN ACUTE HEMORRHAGE AND ULCERATIVE GASTROENTERITIS IN A CHILD. B. A. LAWRENCE, M.D., AND ALLAN BLOXSOM, M.D., HOUSTON, TEXAS 1265

CONGENITAL LEUKEMIA. WESTON M. KELSEY JR., M.D., AND DOROTHY H. ANDERSEN, M.D., NEW YORK 1268

UNUSUAL FATAL PERFORATING WOUND OF THE CHEST. THEODORE J. CURPHEY, M.D., AND A. WALTER FREIREICH, M.D., HEMPSTEAD, N. Y. 1278

PROGRESS IN PEDIATRICS:

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OBITUARIES:

MARTHA WOLLSTEIN, M.D. 1301

ABSTRACTS FROM CURRENT LITERATURE 1302

SOCIETY TRANSACTIONS:

UNIVERSITY OF MICHIGAN PEDIATRIC AND INFECTIOUS DISEASE SOCIETY 1350

NEWS AND COMMENT 1371

NOTICES:

CUMULATED INDEX OF THE AMERICAN JOURNAL OF DISEASES OF CHILDREN 1372

BOOK REVIEWS 1373

DIRECTORY OF PEDIATRIC SOCIETIES 1374

GENERAL INDEX 1381